VTVLDVGDAYFSVPLDEDFR

QUERY	VTVLDVGDAYFSVPLDEDFR
CONSENSUS_A	----------------s--
A.KE.Q23-CXC-CG	--------------H-E--
A.SE.SE6594	--------------H----
A.SE.SE7253	----------------S--
A.SE.SE7535	-----L---------H-G--
A.SE.SE8131	----------------S--
A.SE.SE8538	----------------S--
A.SE.SE8891	---------------KN--
A.UG.92UG037	----------------S--
A.UG.U455	----------------S--
CONSENSUS_B	---------------k---
B.-.NL43E9	---------------K---
B.AU.MBC18	----------------N--
B.AU.MBC200	---------------KE--
B.AU.MBC925	--------------Y----
B.AU.MBCC54	----------------N--
B.AU.MBCC98	---------------K---
B.AU.MBCD36	----------------S--
B.CN.RL42	---------------K---
B.DE.D31	-------------------
B.DE.HAN	---------------K---
B.FR.HXB2	-------------------
B.GA.OYI	---------------K---
B.GB.CAM1	---------------K---
B.GB.MANC	--------------Y----
B.NL.3202A21	G-A-------------K---
B.TW.LM49	---------------K---
B.US.AD8	---------------K---
B.US.BC	--------R-------K---
B.US.DH123	------------I-------
B.US.JRCSF	---------------K---
B.US.JRFL	---------------K---
B.US.MNCG	---------------K---
B.US.NY5CG	---------------K---
B.US.P896	-------------------
B.US.RF	---------------KE--
B.US.SF2	---------------K---
B.US.WEAU160	-------------------
B.US.WR27	---------x---x--xEx-
B.US.YU2	--------------H----
CONSENSUS_C	-------------------
C.BR.92BR025	----------------G--
C.BW.96BW01B03	----------------G--
C.BW.96BW0402	----------------S--
C.BW.96BW0502	-----M----------G--
C.BW.96BW1104	----------------S--
C.BW.96BW1210	----------------S--
C.BW.96BW15B03	-------------------
C.BW.96BW1626	----------------S--
C.BW.96BW17A09	-------------------
C.ET.ETH2220	----------------G--
C.IN.21068	--------------Y----
C.IN.301904	--------------Y----
C.IN.301905	--------------Y----
C.IN.301999	--------------Y----
C.IN.94IN11246	--------------Y----
CONSENSUS_D	-------------------
D.CD.84ZR085	I----------I--C----
D.CD.ELI	-------------------
D.CD.NDK	-------------------
D.CD.Z2Z6	---------------K---
D.UG.94UG1141	---------------H----
CONSENSUS_F1	---------------K---
F1.BE.VI850	---------------K--K
F1.BR.93BR020.1	---------------K---
F1.FI.FIN9363	---------------K---
F1.FR.MP411	---------------KE--
CONSENSUS_F2	---------------KE--
F2.CM.MP255	---------------KE--
F2.CM.MP257	---------------KE--
CONSENSUS_G	-------------------
G.BE.DRCBL	----------------N--
G.FI.HH8793	SA--------------S--
G.NG.92NG083	---------------K---
G.SE.SE6165	-------------------
CONSENSUS_H	-S--------------k---
H.BE.VI991	-S-----G-------H----
H.BE.VI997	-S--------------K---
H.CF.90CF056	-S--------------KE--
CONSENSUS_J	---------------Y----
J.SE.SE9173	---------------Y----
J.SE.SE9280	---------------Y----
CONSENSUS_K	---------------K---
K.CD.EQTB11C	---------------K---
K.CM.MP535	---------------K---
N.CM.YBF30	------------C---K---
CONSENSUS_O	------------C---P---
O.CM.ANT70C	------------C---P---
O.CM.MVP5180	------------C---P---
AC.ET.E3099G	----------------S--
AC.IN.21301	---------------Y-E--
AC.RW.92RW009	----------------S--
AC.SE.SE9488	---------------K---
AC.ZM.ZAM184	---------------H----
ACD.SE.SE8603	--I--------------S--
AD.SE.SE6954	---------------Y----
AD.SE.SE7108	---------------H-E--
ADU.CD.MAL	-------------------
AG.NG.G3	------------I----N--
AG.SE.SE7812	---------------K---
AGHU.GA.VI354	---------------Y-G--
AGHU.NO.NOGIL3	---------------A----
AGJ.AU.BFP90	------------I----K--
AGJ.ML.95ML8	---------------G-N--
AGU.CD.Z321	----------------H----
BF.BR.93BR029.4	----------------K---
CRF01_AE.CF.90CF40	-----------------G--
CRF01_AE.TH.93TH25	-----------------S--
CRF01_AE.TH.CM240	-----------------S--
CRF01_AE.TH.TH022	-----------------S--
CRF01_AE.TH.TH047	-----------------S--
CRF02_AG.FR.DJ263	----------------K---
CRF02_AG.FR.DJ264	----------------K---
CRF02_AG.NG.IBNG	----------------K---
CRF03_AB.RU.KAL153	----------------Q---
CRF04_CPX.CY.94CY0	----------------PE--
CRF04_CPX.GR.97PVC	----------------PA--
CRF04_CPX.GR.97PVM	----------------PE--
DF.CD.VI961	---------------H----
U.CD.VI1126	----------------PE--
CONSENSUS_CPZ	------------c---k---
CPZ.CD.CPZANT	------------I---Q---
CPZ.GA.CPZGAB	------------C---K---
CPZ.US.CPZUS	------------C---KE--

KYTAFTIPSINNETPGIRYQ

QUERY	KYTAFTIPSINNETPGIRYQ
CONSENSUS_A	---------t----------
A.KE.Q23-CXC-CG	---------T------V---
A.SE.SE6594	---------T--A-------
A.SE.SE7253	---------T------V---
A.SE.SE7535	---------T--A-------
A.SE.SE8131	---------T----------
A.SE.SE8538	---------T----------
A.SE.SE8891	--------------------
A.UG.92UG037	---------T----------
A.UG.U455	----------------V---
CONSENSUS_B	--------------------
B.-.NL43E9	--------------------
B.AU.MBC18	--------------------
B.AU.MBC200	--------------------
B.AU.MBC925	---------T----------
B.AU.MBCC54	--------------------
B.AU.MBCC98	--------------------
B.AU.MBCD36	----------------T---
B.CN.RL42	---------V----------
B.DE.D31	----------------V---
B.DE.HAN	--------------------
B.FR.HXB2	--------------------
B.GA.OYI	--------------------
B.GB.CAM1	---------T----------
B.GB.MANC	---V---------A--V---
B.NL.3202A21	---------V----------
B.TW.LM49	--------------------
B.US.AD8	--------------------
B.US.BC	--------------------
B.US.DH123	---------V--AA------
B.US.JRCSF	--------------------
B.US.JRFL	--------------------
B.US.MNCG	--------------------
B.US.NY5CG	--------------------
B.US.P896	--------------------
B.US.RF	---------------R----
B.US.SF2	--------------------
B.US.WEAU160	--------------------
B.US.WR27	---------xH---------
B.US.YU2	----------------T---
CONSENSUS_C	--------------------
C.BR.92BR025	--------------------
C.BW.96BW01B03	--------------------
C.BW.96BW0402	------------S-------
C.BW.96BW0502	--------------------
C.BW.96BW1104	--------------------
C.BW.96BW1210	---------R----------
C.BW.96BW15B03	--------------------
C.BW.96BW1626	------------A-------
C.BW.96BW17A09	--------------------
C.ET.ETH2220	---------T----------
C.IN.21068	--------------------
C.IN.301904	--------------------
C.IN.301905	---------V----------
C.IN.301999	---------R----------
C.IN.94IN11246	--------------------
CONSENSUS_D	--------------------
D.CD.84ZR085	--------------------
D.CD.ELI	-------S------------
D.CD.NDK	--------------------
D.CD.Z2Z6	--------------------
D.UG.94UG1141	---------T----------
CONSENSUS_F1	---------v----------
F1.BE.VI850	---------V----------
F1.BR.93BR020.1	----S----T------V---
F1.FI.FIN9363	---------V----------
F1.FR.MP411	---------L----------
CONSENSUS_F2	--------------------
F2.CM.MP255	--------------------
F2.CM.MP257	--------------------
CONSENSUS_G	---------?----------
G.BE.DRCBL	---------T----------
G.FI.HH8793	---------T----------
G.NG.92NG083	--------------------
G.SE.SE6165	----------------V---
CONSENSUS_H	--------------------
H.BE.VI991	---------T----------
H.BE.VI997	--------------------
H.CF.90CF056	--------------------
CONSENSUS_J	--------------------
J.SE.SE9173	--------------------
J.SE.SE9280	--------------------
CONSENSUS_K	--------------------
K.CD.EQTB11C	--------------------
K.CM.MP535	----------------V---
N.CM.YBF30	--------------------
CONSENSUS_O	---------V------?---
O.CM.ANT70C	---------V----------
O.CM.MVP5180	---------V------V---
AC.ET.E3099G	--------------------
AC.IN.21301	---------T----------
AC.RW.92RW009	--------------------
AC.SE.SE9488	---------T----------
AC.ZM.ZAM184	--------------------
ACD.SE.SE8603	---------T----------
AD.SE.SE6954	--------------------
AD.SE.SE7108	---------T----------
ADU.CD.MAL	--------------------
AG.NG.G3	--------------------
AG.SE.SE7812	--------------------
AGHU.GA.VI354	R-------------------
AGHU.NO.NOGIL3	--------------------
AGJ.AU.BFP90	---------T----------
AGJ.ML.95ML8	---------L----------
AGU.CD.Z321	--------------------
BF.BR.93BR029.4	---------T------L---
CRF01_AE.CF.90CF40	----------------V---
CRF01_AE.TH.93TH25	--------------------
CRF01_AE.TH.CM240	--------------------
CRF01_AE.TH.TH022	--------------------
CRF01_AE.TH.TH047	--------------------
CRF02_AG.FR.DJ263	---------T----------
CRF02_AG.FR.DJ264	---------V----------
CRF02_AG.NG.IBNG	---------V----------
CRF03_AB.RU.KAL153	---------T----------
CRF04_CPX.CY.94CY0	---------T----------
CRF04_CPX.GR.97PVC	---------T------V---
CRF04_CPX.GR.97PVM	--------------------
DF.CD.VI961	----------------F---
U.CD.VI1126	---------V------V---
CONSENSUS_CPZ	---------v----------
CPZ.CD.CPZANT	---------V---------C
CPZ.GA.CPZGAB	----------------V---
CPZ.US.CPZUS	---------V----------

HPAGLKKKKSVTVLDVGDAY

QUERY	HPAGLKKKKSVTVLDVGDAY
CONSENSUS_A	--------------------
A.KE.Q23-CXC-CG	--------------------
A.SE.SE6594	--------N----------
A.SE.SE7253	--------------------
A.SE.SE7535	---------------L----
A.SE.SE8131	--------------------
A.SE.SE8538	--------------------
A.SE.SE8891	----$---------------
A.UG.92UG037	--------------------
A.UG.U455	-T------------------
CONSENSUS_B	--------------------
B.-.NL43E9	------Q-------------
B.AU.MBC18	--S-----R-----------
B.AU.MBC200	--------------------
B.AU.MBC925	--------------------
B.AU.MBCC54	--------------------
B.AU.MBCC98	--------------------
B.AU.MBCD36	--S-----------------
B.CN.RL42	--------------------
B.DE.D31	--------------------
B.DE.HAN	--------------------
B.FR.HXB2	--------------------
B.GA.OYI	--------------------
B.GB.CAM1	--------------------
B.GB.MANC	--------------------
B.NL.3202A21	----------G-A-------
B.TW.LM49	--------------------
B.US.AD8	--------------------
B.US.BC	------------------R-
B.US.DH123	--------------------
B.US.JRCSF	--------------------
B.US.JRFL	-------R------------
B.US.MNCG	--------------------
B.US.NY5CG	------Q-------------
B.US.P896	--------------------
B.US.RF	--------------------
B.US.SF2	--------------------
B.US.WEAU160	--S-----------------
B.US.WR27	-------------------x
B.US.YU2	--------------------
CONSENSUS_C	--------------------
C.BR.92BR025	--------------------
C.BW.96BW01B03	--------------------
C.BW.96BW0402	--------------------
C.BW.96BW0502	---------------M----
C.BW.96BW1104	--------------------
C.BW.96BW1210	--------------------
C.BW.96BW15B03	--------------------
C.BW.96BW1626	--------------------
C.BW.96BW17A09	----S---------------
C.ET.ETH2220	--------------------
C.IN.21068	--------------------
C.IN.301904	--------------------
C.IN.301905	--------------------
C.IN.301999	--------------------
C.IN.94IN11246	--------------------
CONSENSUS_D	--------------------
D.CD.84ZR085	----------I---------
D.CD.ELI	--------------------
D.CD.NDK	--------------------
D.CD.Z2Z6	--------------------
D.UG.94UG1141	--------------------
CONSENSUS_F1	--------------------
F1.BE.VI850	--------------------
F1.BR.93BR020.1	--------------------
F1.FI.FIN9363	--------------------
F1.FR.MP411	--------------------
CONSENSUS_F2	--------?-----------
F2.CM.MP255	--------------------
F2.CM.MP257	--------R-----------
CONSENSUS_G	--------r-----------
G.BE.DRCBL	--G---Q-R-----------
G.FI.HH8793	------RENQSA--------
G.NG.92NG083	--------R-----------
G.SE.SE6165	--------------------
CONSENSUS_H	-----------S--------
H.BE.VI991	-----------S-----G--
H.BE.VI997	-----------S--------
H.CF.90CF056	-----------S--------
CONSENSUS_J	--------------------
J.SE.SE9173	--------------------
J.SE.SE9280	--------------------
CONSENSUS_K	--------------------
K.CD.EQTB11C	--------------------
K.CM.MP535	--------------------
N.CM.YBF30	------Q-------------
CONSENSUS_O	--G---Q?Q-----------
O.CM.ANT70C	--G---Q-Q-----------
O.CM.MVP5180	--G---QRQ-----------
AC.ET.E3099G	--------R-----------
AC.IN.21301	--------------------
AC.RW.92RW009	--------------------
AC.SE.SE9488	--------------------
AC.ZM.ZAM184	--------------------
ACD.SE.SE8603	------------I-------
AD.SE.SE6954	--------------------
AD.SE.SE7108	--------------------
ADU.CD.MAL	--------------------
AG.NG.G3	--------R-----------
AG.SE.SE7812	--------------------
AGHU.GA.VI354	--------------------
AGHU.NO.NOGIL3	--------------------
AGJ.AU.BFP90	--------------------
AGJ.ML.95ML8	-------R------------
AGU.CD.Z321	---R----R-----------
BF.BR.93BR029.4	--------------------
CRF01_AE.CF.90CF40	--------------------
CRF01_AE.TH.93TH25	--------------------
CRF01_AE.TH.CM240	--------------------
CRF01_AE.TH.TH022	R-------------------
CRF01_AE.TH.TH047	--------------------
CRF02_AG.FR.DJ263	--------------------
CRF02_AG.FR.DJ264	--------------------
CRF02_AG.NG.IBNG	--------------------
CRF03_AB.RU.KAL153	--------------------
CRF04_CPX.CY.94CY0	--------------------
CRF04_CPX.GR.97PVC	--------------------
CRF04_CPX.GR.97PVM	--------------------
DF.CD.VI961	--------------------
U.CD.VI1126	--------------------
CONSENSUS_CPZ	--------------------
CPZ.CD.CPZANT	------Q-------------
CPZ.GA.CPZGAB	--------------------
CPZ.US.CPZUS	--------R-----------

Study Subject ID:00RCH34
Study Subject Clone:
Study Subject HLA:A31,A36,B64,B35,Cw4,Cw8
Sequence: Known reactive 20Mer0: VTVLDVGDAYFSVPLDEDFR RT(106–125)

Possible HLA

A31	A*3101,A*3104,A*3201,A*3202
A36	A*3601
B35	B*35,B*1522,B*3501,B*3502,B*3503,B*3504,B*3505,B*3506,B*3507,B*3508,B*3509,B*3511,B*3512,B*3513,B*3514,B*3515,B*3517,B*3518,B*3519,B*3520
B64	B*1401
Cw4	C4,Cw*0401,C*0401,Cw*0402
Cw8	Cw*08,Cw*0801,Cw*0802,C*0802,Cw*0803

Possible Epitopes based on anchor residues

(12-20)	SVPLDEDFR	A*3101
(13-20)	VPLDEDFR	A*3101
(11-20)	FSVPLDEDFR	A*3101
(10-19)	YFSVPLDEDF	Cw*0401

Anchor Residues Searched

A*3101	XXXXXXXX[R]
A*3101	XXXXXXX[R]
A*3101	XXXXXXXXX[R]
B*35	X[P]XXXXXX[YFMLI]
B*35	X[P]XXXXX[YFMLI]
B*35	X[P]XXXXXXX[YFMLI]
B*3501	X[P]XXXXXX[YFMLI]
B*3501	X[P]XXXXX[YFMLI]
B*3501	X[P]XXXXXXX[YFMLI]
B*3503	X[P]XXXXXX[M]
B*3503	X[P]XXXXX[M]
B*3503	X[P]XXXXXXX[M]
Cw*0401	X[YPF]XXXXXX[LF]
Cw*0401	X[YPF]XXXXX[LF]
Cw*0401	X[YPF]XXXXXXX[LF]

Study Subject ID:00RCH34
Study Subject Clone:
Study Subject HLA:A31,A36,B64,B35,Cw4,Cw8
Sequence: Known reactive 20Mer1: KYTAFTIPSINNETPGIRYQ RT(126–145)

Possible HLA

A31	A*3101,A*3104,A*3201,A*3202
A36	A*3601
B35	B*35,B*1522,B*3501,B*3502,B*3503,B*3504,B*3505,B*3506,B*3507,B*3508,B*3509,B*3511,B*3512,B*3513,B*3514,B*3515,B*3517,B*3518,B*3519,B*3520
B64	B*1401
Cw4	C4,Cw*0401,C*0401,Cw*0402
Cw8	Cw*08,Cw*0801,Cw*0802,C*0802,Cw*0803

Possible Epitopes based on anchor residues

(10-18)	INNETPGIR	A*3101
(11-18)	NNETPGIR	A*3101
(9-18)	SINNETPGIR	A*3101

Anchor Residues Searched

A*3101	XXXXXXXX[R]
A*3101	XXXXXXX[R]
A*3101	XXXXXXXXX[R]
B*35	X[P]XXXXXX[YFMLI]
B*35	X[P]XXXXX[YFMLI]
B*35	X[P]XXXXXXX[YFMLI]
B*3501	X[P]XXXXXX[YFMLI]
B*3501	X[P]XXXXX[YFMLI]
B*3501	X[P]XXXXXXX[YFMLI]
B*3503	X[P]XXXXXX[M]
B*3503	X[P]XXXXX[M]
B*3503	X[P]XXXXXXX[M]
Cw*0401	X[YPF]XXXXXX[LF]
Cw*0401	X[YPF]XXXXX[LF]
Cw*0401	X[YPF]XXXXXXX[LF]

Study Subject ID:00RCH34
Study Subject Clone:
Study Subject HLA:A31,A36,B64,B35,Cw4,Cw8
Sequence: Known reactive 20Mer2: HPAGLKKKKSVTVLDVGDAY RT(96–115)

Possible HLA

A31	A*3101,A*3104,A*3201,A*3202
A36	A*3601
B35	B*35,B*1522,B*3501,B*3502,B*3503,B*3504,B*3505,B*3506,B*3507,B*3508,B*3509,B*3511,B*3512,B*3513,B*3514,B*3515,B*3517,B*3518,B*3519,B*3520
B64	B*1401
Cw4	C4,Cw*0401,C*0401,Cw*0402
Cw8	Cw*08,Cw*0801,Cw*0802,C*0802,Cw*0803

Possible Epitopes based on anchor residues

Anchor Residues Searched

A*3101	XXXXXXXX[R]
A*3101	XXXXXXX[R]
A*3101	XXXXXXXXX[R]
B*35	X[P]XXXXXX[YFMLI]
B*35	X[P]XXXXX[YFMLI]
B*35	X[P]XXXXXXX[YFMLI]
B*3501	X[P]XXXXXX[YFMLI]
B*3501	X[P]XXXXX[YFMLI]
B*3501	X[P]XXXXXXX[YFMLI]
B*3503	X[P]XXXXXX[M]
B*3503	X[P]XXXXX[M]
B*3503	X[P]XXXXXXX[M]
Cw*0401	X[YPF]XXXXXX[LF]
Cw*0401	X[YPF]XXXXX[LF]
Cw*0401	X[YPF]XXXXXXX[LF]

This table lists epitopes that are experimentally observed to be presented by a HLA type carried by the patient, but the de£ned epitope has substitutions relative to the peptides from your reference strains and so might be missed by your reagents: in HXB2 for Gag, Pol; MN for Env; BRU for Nef, relative to most B clade Sequences in the database:

Protein	Epitope in Database	Epitope in Ref. strain	Epitope in Consensus B	HLA	Notes
p17(124–132)	NSSKVSQNY	HSNQVSQNY	NSSQVSQNY	B*3501	
p17(124–132)	NSSKVSQNY	HSNQVSQNY	NSSQVSQNY	B35	
p24(122–130)	PPIPVGDIY	PPIPVGEIY	PPIPVGEIY	B*3501	
p24(122–130)	NPVPVGNIY	PPIPVGEIY	PPIPVGEIY	B*3501	
p24(122–130)	PPIPVGDIY	PPIPVGEIY	PPIPVGEIY	B35	
p24(122–130)	PPIPVGDIY	PPIPVGEIY	PPIPVGEIY	B35	
RT(118–127)	VPLDKDFRKY	VPLDEDFRKY	VPLDKDFRKY	B*3501	
RT(118–127)	VPLDKDFRKY	VPLDEDFRKY	VPLDKDFRKY	B35	
RT(175–183)	HPDIVIYQY	NPDIVIYQY	NPDIVIYQY	B*3501	
RT(175–183)	HPDIVIYQY	NPDIVIYQY	NPDIVIYQY	B35	
RT(175–183)	HPDIVIYQY	NPDIVIYQY	NPDIVIYQY	B35	
RT(175–183)	HPDIVIYQY	NPDIVIYQY	NPDIVIYQY	B35	
gp160(78–86)	DPNPQEVVL	DPNPQEVEL	DPNPQEVVL	B*3501	
gp160(78–86)	DPNPQEVVL	DPNPQEVEL	DPNPQEVVL	B35	
gp160(78–86)	DPNPQEVVL	DPNPQEVEL	DPNPQEVVL	B35, B51	
gp160(156–165)	NCSFNISTSI	NCSFNITTSI	NCSFNITTSI	Cw*08	
gp160(156–165)	NCSFNISTSI	NCSFNITTSI	NCSFNITTSI	Cw8	
gp160(239–247)	CTNVSTVQC	CKNVSTVQC	CTNVSTVQC	Cw8	
gp160(252–260)	RPIVSTQLL	RPVVSTQLL	RPVVSTQLL	B*3501	
gp160(252–260)	RPIVSTQLL	RPVVSTQLL	RPVVSTQLL	B35	
gp160(419–427)	RIKQIINMW	KIKQIINMW	RIKQIINMW	A*3201	
gp160(606–614)	TAVPWNASW	TTVPWNASW	TAVPWNASW	B*3501	
gp160(606–614)	TAVPWNASW	TTVPWNASW	TAVPWNASW	B35	
gp160(770–780)	RLRDLLLIVTR	HHRDLLLIAAR	RLRDLLLIVTR	A*3101	
gp160(770–780)	RLRDLLLIVTR	HHRDLLLIAAR	RLRDLLLIVTR	A31	
Nef(68–76)	FPVRPQVPL	FPVTPQVPL	FPVRPQVPL	B*3501	
Nef(68–76)	FPVRPQVPL	FPVTPQVPL	FPVRPQVPL	B35	
Nef(69–79)	RPQVPLRPMTY	TPQVPLRPMTY	RPQVPLRPMTY	B35	
Nef(71–81)	RPQVPLRPMTY	TPQVPLRPMTY	RPQVPLRPMTY	B*3501	
Nef(71–81)	RPQVPLRPMTY	TPQVPLRPMTY	RPQVPLRPMTY	B35	
Nef(73–82)	SVPLRPMTYK	QVPLRPMTYK	QVPLRPMTYK	B35 or C4	
Nef(135–143)	YPLTFGWCF	YPLTFGWCY	YPLTFGWCF	B35	

Table 1: **p17**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p17(124–132)	p17(124–132 LAI)	NSSKVSQNY	HIV-1 or -2 infection	human(B*3501)	[Brander & Goulder(2001)]
	• Noted by Brander to be B*3501 epitope				
p17(124–132)	p17(124–132 LAI)	NSSKVSQNY	HIV-1 infection	human(B35)	[McMichael & Walker(1994)]
	• Review of HIV CTL epitopes				

Table 2: **p24**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
p24(122–130)	p24(260–268 LAI)	PPIPVGDIY	HIV-1 or -2 infection	human(B*3501)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*3501 epitope				
p24(122–130)	p24(245–253 HIV-2)	NPVPVGNIY	HIV-1 infection	human(B*3501)	[Rowland-Jones (1995)]
p24(122–130)	p24(260–268 LAI)	PPIPVGDIY	HIV-1 or -2 infection	human(B35)	[Rowland-Jones (1995)]
	• De£ned as minimal peptide by titration curve, PPIPVGEIY and HIV-2 form NPVPVGNIY are also recognized				
p24(122–130)	p24()	PPIPVGDIY		human(B35)	[Rowland-Jones (1999)]
	• CTL responses in seronegative highly HIV-exposed African female sex workers in Gambia and Nairobi were studied – these women had no delta 32 deletion in CCR5 • In Gambia there is exposure to both HIV-1 and HIV-2, CTL responses to B35 epitopes in exposed, uninfected women are cross-reactive, and the B35 allele seems to be protective • HIV-2 version of this epitope is not conserved: NPVPVGNIY, but the CTLs are cross-reactive – one of £ve B35 CTL epitopes that are cross-reactive, see also [Rowland-Jones (1995)]				

Table 3: **RT**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
RT(118–127)	RT(273–282 SF2)	VPLDKDFRKY	HIV-1 infection	human(B*3501)	[Tomiyama (1997), Menendez-Arias (1998)]
	• A CTL clone responsive to this epitope was obtained • 4/7 B35-positive individuals had a CTL response to this epitope • A K to E substitution at position 5 abrogates speci£c lysis, and reduces binding to B*3501 • [Menendez-Arias (1998)], in a review, notes that a Glu to Lys (E to K) change abrogates CTL activity, but that both VPLDEDFRKY and VPLDKDFRKY can serve as HLA-B35 epitopes, so the change must alter T cell receptor binding – residues in this epitope may be important for polymerase activity				
RT(118–127)	()	VPLDKDFRKY	HIV-1 infection	human(B35)	[Kawana (1999)]
	• HLA B35 is associated with rapid disease progression • The sequences of 9 previously described HIV-1 B35 CTL epitopes were obtained in 10 HLA B35+ and 19 HLA B35- individuals • 3/9 CTL epitopes had substitutions that were more common in B35+ individuals than in B35- individuals – only one of these reduced the binding of the peptide to B35 and was shown to be an escape mutation • ––E—- was found in 8/10 of the B35+ individuals, and three of the B35- individuals – the D –> E substituted peptide had similar binding af£nity to B35 and was equally susceptible to a CTL clone				
RT(175–183)	RT(342–350 LAI)	HPDIVIYQY	HIV-1 infection	human(B*3501)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*3501 epitope				
RT(175–183)	RT(342–350 LAI)	HPDIVIYQY	HIV-1 infection	human(B35)	[McMichael & Walker(1994)]
	• Review of HIV CTL epitopes				
RT(175–183)	RT(329–337)	HPDIVIYQY	none	human(B35)	[Lalvani (1997)]
	• A peptide-based protocol was optimized for restimulation of CTLp using optimized peptide and IL-7 concentrations – importantly this protocol does not stimulate a primary response, only secondary – peptide-speci£c CTLp counts could be obtained via staining with peptide-Class I tetramers • This peptide was one of the B35 presented test peptides used in control experiments showing that the assay gave no activity using lymphocytes from 21 healthy B35 seronegative donors				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
RT(175–183)	Pol()	HPDIVIYQY		human(B35)	[Rowland-Jones (1999)]

- CTL responses in seronegative highly HIV-exposed African female sex workers in Gambia and Nairobi were studied – these women had no delta 32 deletion in CCR5
- In Gambia there is exposure to both HIV-1 and HIV-2, CTL responses to B35 epitopes in exposed, uninfected women are cross-reactive, and the B35 allele seems to be protective
- HIV-2 version of this epitope is not conserved: NPDVILIQY, but the CTLs are cross-reactive – one of £ve B35 CTL epitopes that are cross-reactive, see also [Rowland-Jones (1995)]

Table 4: **gp160**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(78–86)	gp120(77–85)	DPNPQEVVL	HIV-1 infection	human(B*3501)	[Ogg (1998)]
	• This epitope was included to illustrate the speci£city of HIV-tetrameric staining, in a cross-sectional study correlating HLA A*0201 CTL effector cells and low viral load				
gp160(78–86)	Env(77–85)	DPNPQEVVL	HIV-1 infection	human(B35)	[Dyer (1999)]
	• CTL speci£c responses were measured over a 1.3 to 1.5 year period in members of the Sydney Blood Bank Cohort (SBBC) who had been infected with a natural attenuated strain of HIV-1 which was Nef-defective • Some of these patients had prolonged high levels of CTL effector and memory cells despite low viral load				
gp160(78–86)	gp120(77–85 SF2)	DPNPQEVVL	HIV-1 infection	human(B35, B51)	[Shiga (1996)]
	• Binds HLA-B*3501 and B*5101 – binds and kills gp120-vaccinia virus infected cells carrying B35 or B51				
gp160(156–165)	gp120(156–165)	NCSFNISTSI	HIV-1 infection	human(Cw*08)	[Ferris (1999)]
	• Recognized by CTL clone LWF A5, isolated from a lab worker exposed to HIV-1 in 1985 • The processing of this epitope is TAP1/2-dependent, as are most Env epitopes, and it contains two N-linked glycosylation sites that are glycosylated in Env • Only peptide that has been deglycosylated, a process that changes asparagine (N) to aspartic acid (D) was recognized: the aspartic acid at position 5 was critical, position 1 could be either D or N • This peptide also contains a Cys involved in a disul£de linkage but reducing conditions did not effect recognition by CTL clone LWF A5 • The HIV-1 Env epitopes are typically processed by a TAP1/2 dependent mechanism, which involves cotranslational translocation into the ER, glycosylation, export back into the cytosol, and deglycosylation for processing, and retransport into the ER for the association with class I molecules • The particular pathway of generating an epitope may have an impact on the presentation of that epitope, quantitatively as well as qualitatively				
gp160(156–165)	gp120(156–165 IIIB)	NCSFNISTSI	HIV-1 infection	human(Cw8)	[Sipsas (1997)]
	• HIV IIIB proteins were used to de£ne the range of CTL epitopes recognized by 3 lab workers accidentally infected with HIV-1 IIIB • NCSFNITTSI, a variant found in HIV-1 MN, was not recognized, thus this epitope was type-speci£c • NCSFNISTSI contains two potential N-linked glycosylation sites and cysteine residue, possibly related to the requirement for a high sensitizing dose of peptide for CTL activity				
gp160(239–247)	gp120(241–249 LAI)	CTNVSTVQC	HIV-1 infection	human(Cw8)	[Sipsas (1997)]
	• HIV IIIB proteins were used to de£ne the range of CTL epitopes recognized by 3 lab workers accidentally infected with HIV-1 IIIB • CTNVSTVQC contains a potential N-linked glycosylation site and cysteine residues, possibly related to a requirement for a high sensitizing dose of peptide for CTL activity				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
gp160(252–260)	gp120(255–263 SF2)	RPIVSTQLL	HIV-1 infection	human(B*3501)	[Tomiyama (1997)]
	• A CTL clone responsive to this epitope was obtained • Only 1/7 B35-positive individuals had a CTL response to this epitope • An I to V substitution at position 3 reduces speci£c lysis, but not binding to B*3501 • A Q to H substitution at position 7 abrogates speci£c lysis, but not binding to B*3501				
gp160(252–260)	gp120(255–263 SF2)	RPIVSTQLL	HIV-1 infection	human(B35)	[Shiga (1996)]
	• Binds HLA-B*3501				
gp160(419–427)	gp120(424–432 HXB2)	RIKQIINMW		human(A*3201)	[Harrer (1996)]
	• C. Brander notes that this is an A*3201 epitope in the 1999 database				
gp160(606–614)	gp41(605–615 LAI)	TAVPWNASW	gp160 vaccinia	human(B*3501)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*3501 epitope				
gp160(606–614)	gp41(605–615 LAI)	TAVPWNASW	gp160 vaccinia	human(B35)	[Johnson (1994)]
	• Epitope for vaccine induced CD8+ clone				
gp160(770–780)	gp41(770–780 BH10)	RLRDLLLIVTR	HIV-1 infection	human(A*3101)	[Safrit (1994a), Safrit (1994b)]
	• Recognized by CTL derived from acute seroconverter • C. Brander notes that this is an A*3101 epitope in the 1999 database				
gp160(770–780)	gp41(770–780)	RLRDLLLIVTR	HIV-1 infection	human(A31)	[Ferris (1999), Hammond (1995)]
	• This epitope is processed by a TAP1/2 dependent mechanism				

Table 5: **Nef**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
Nef(68–76)	Nef(72–80 SF2)	FPVRPQVPL	HIV-1 infection	human(B*3501)	[Tomiyama (1997)]
	• A CTL clone responsive to this epitope was obtained • 3/7 B35-positive individuals had a CTL response to this epitope • An R to T substitution at position 4 abrogates speci£c lysis, but not binding to B*3501				
Nef(68–76)	Nef(72–80 SF2)	FPVRPQVPL	HIV-1 infection	human(B35)	[Shiga (1996)]
	• Binds HLA-B*3501				
Nef(69–79)	()	RPQVPLRPMTY	HIV-1 infection	human(B35)	[Kawana (1999)]
	• HLA B35 is associated with rapid disease progression • The sequences of 9 previously described HIV-1 B35 CTL epitopes were obtained in 10 HLA B35+ and 19 HLA B35- individuals • 3/9 CTL epitopes had substitutions that were more common in B35+ individuals than in B35- individuals – only one of these reduced the binding of the peptide to B35 and was shown to be an escape mutation • ————-F was found in 9/10 of the B35+ individuals, none of the B35- individuals – the Y –> F substituted peptide had a similar binding af£nity with B35 and was recognized by a CTL clone equally with wildtype				
Nef(71–81)	Nef(75–85 SF2)	RPQVPLRPMTY	HIV-1 infection	human(B*3501)	[Tomiyama (1997)]
	• A CTL clone responsive to this epitope was obtained • 4/7 B35-positive individuals had a strong CTL response to this epitope • An R to T substitution at position 1 abrogates speci£c lysis, but not binding to B*3501 • An R to H substitution at position 7 did not alter reactivity				
Nef(71–81)	Nef(75–85 SF2)	RPQVPLRPMTY	HIV-1 infection	human(B35)	[Shiga (1996)]
	• Binds HLA-B*3501				
Nef(73–82)	Nef(73–82 LAI)	SVPLRPMTYK	HIV-1 infection	human(B35 or C4)	[Buseyne (1993)]
	• Vertical transmission of HIV ranges from 13% to 39% • Primary assays showed cytotoxic activity against at least one HIV protein was detected in 70% of infected children • Epitopes recognized in £ve children were mapped using synthetic peptides and secondary cultures • Patient EM13, who had a CTL response to three epitopes in Nef, was infected via blood transfusion after birth and went from CDC stage P2A to P2E during the study				
Nef(135–143)	Nef(139–147 SF2)	YPLTFGWCF	HIV-1 infection	human(B35)	[Shiga (1996)]
	• Binds HLA-B*3501				

Table 6: **All De£ned Epitopes within the 20mer, regardless of HLA type**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
RT(107–115)	RT(262–270 IIIB)	TVLDVGDAY		(B*3501)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*3501 epitope				
RT(107–115)	RT(262–270 IIIB)	TVLDVGDAY	HIV-1 infection	human(B35)	[Wilson (1996), Menendez-Arias (1998)]
	• Epitope de£ned in the context of the Pediatric AIDS Foundation ARIEL Project, a mother-infant HIV transmission study • TVLDMGDAC is a naturally occurring variant that is less reactive • [Menendez-Arias (1998)], in a review, notes that this epitope includes a catalytic residue (Asp-110) in the active site of RT				
RT(107–115)	Pol(262–270 IIIB)	TVLDVGDAY	HIV-1 infection	human(B35)	[Wilson (1999)]
	• This study describes maternal CTL responses in the context of mother-to-infant transmission • Detection of CTL escape mutants in the mother was associated with transmission, but the CTL-susceptible forms of the virus tended to be found in infected infants • An additional variant that gave a positive CTL response: TVLDMGDAC				
RT(108–118)	RT(267–277)	VLDVGDAYFSV	*in vitro* stimulation	human(A*0201)	[van der Burg (1996)]
	• High dissociation rate, but immunogenic in primary CTL induction after repeated stimulations with peptide • CTL generated by *in vitro* stimulation of PBMC derived from uninfected individual				
RT(108–118)	RT(267–277)	VLDVGDAYFSV	HIV-1 infection	human(A2)	[Kundu (1998)]
	• Allogeneic dendritic cells (DCs) were obtained from HLA-identical siblings, pulsed with rgp160 MN or A2-restricted HIV-1 epitope peptides, and infused monthly into six HIV-infected patients • 1/6 showed increased env-speci£c CTL and increased lymphoproliferative responses, 2/6 showed increase only in proliferative responses, and 3/6 showed no change – pulsed DCs were well tolerated • VLDVGDAYFSV is a conserved HLA-A2 epitope included in this study – 4/6 patients had this sequence as their HIV direct sequence, but only one of these had a detectable CTL response – the other two had the sequences EEDVGDAYFSV and ELDVGDAYFSV and no detectable CTL response				
RT(108–118)	RT(267–277)	VLDVGDAYFSV	*in vitro* stimulation	human(A2)	[van der Burg (1995)]
	• Binds HLA-A*0201 – CTL generated by *in vitro* stimulation of PBMC from an HIV negative donor • VLDVGDAYFSV is in a functional domain				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
RT(108–122)	RT(257–251)	VLDVGDAYFSVPLDE	HIV-1 infection	human(Cw4)	[Bernard (1998)]
	• This study focuses on six rare long-term survivor HIV-infected people who were infected for many years without exhibiting immune dysregulation – such immunologically normal HIV-infected (INHI) cases occur at a frequency between 0.1 and 1% in the infected population • No direct CTL were found in any of the six INHIs, but above background CTLp activity was founded in 3/6 INHIs				
RT(113–120)	Pol(268–275 SF2)	DAYFSVPL	HIV-1 infection	human(B*5101, B24)	[Tomiyama (1999)]
	• HLA-B27, -B51, and -B57 are associated with slow progression to AIDS, while HLA -B35, -B8, -B24 are associated with a rapid progression to AIDS (Nat. Med. 2:405, 1996;Lancet 22:1187, 1986;Hum Immunol 22:73, 1988;Hum Immunol 44:156, 1995) • 15% of Japanese populations carry HLA-B51 while HLA-B27 and -B57 are detected in less than 0.3% • Of the 172 HIV-1 peptides with HLA-B*5101 anchor residues, 33 bound to HLA-B*5101, seven of these peptides were reactive with CTL from 3 B*5101 positive individuals, and six were properly processed • Four of the six epitopes were highly conserved among B subtype sequences, DAYFSVPL is conserved				

Table 7: **All De£ned Epitopes within the 20mer, regardless of HLA type**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
RT(107–115)	RT(262–270 IIIB)	TVLDVGDAY		(B*3501)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*3501 epitope				
RT(107–115)	RT(262–270 IIIB)	TVLDVGDAY	HIV-1 infection	human(B35)	[Wilson (1996), Menendez-Arias (1998)]
	• Epitope de£ned in the context of the Pediatric AIDS Foundation ARIEL Project, a mother-infant HIV transmission study • TVLDMGDAC is a naturally occurring variant that is less reactive • [Menendez-Arias (1998)], in a review, notes that this epitope includes a catalytic residue (Asp-110) in the active site of RT				
RT(107–115)	Pol(262–270 IIIB)	TVLDVGDAY	HIV-1 infection	human(B35)	[Wilson (1999)]
	• This study describes maternal CTL responses in the context of mother-to-infant transmission • Detection of CTL escape mutants in the mother was associated with transmission, but the CTL-susceptible forms of the virus tended to be found in infected infants • An additional variant that gave a positive CTL response: TVLDMGDAC				
RT(108–118)	RT(267–277)	VLDVGDAYFSV	*in vitro* stimulation	human(A*0201)	[van der Burg (1996)]
	• High dissociation rate, but immunogenic in primary CTL induction after repeated stimulations with peptide • CTL generated by *in vitro* stimulation of PBMC derived from uninfected individual				
RT(108–118)	RT(267–277)	VLDVGDAYFSV	HIV-1 infection	human(A2)	[Kundu (1998)]
	• Allogeneic dendritic cells (DCs) were obtained from HLA-identical siblings, pulsed with rgp160 MN or A2-restricted HIV-1 epitope peptides, and infused monthly into six HIV-infected patients • 1/6 showed increased env-speci£c CTL and increased lymphoproliferative responses, 2/6 showed increase only in proliferative responses, and 3/6 showed no change – pulsed DCs were well tolerated • VLDVGDAYFSV is a conserved HLA-A2 epitope included in this study – 4/6 patients had this sequence as their HIV direct sequence, but only one of these had a detectable CTL response – the other two had the sequences EEDVGDAYFSV and ELDVGDAYFSV and no detectable CTL response				
RT(108–118)	RT(267–277)	VLDVGDAYFSV	*in vitro* stimulation	human(A2)	[van der Burg (1995)]
	• Binds HLA-A*0201 – CTL generated by *in vitro* stimulation of PBMC from an HIV negative donor • VLDVGDAYFSV is in a functional domain				

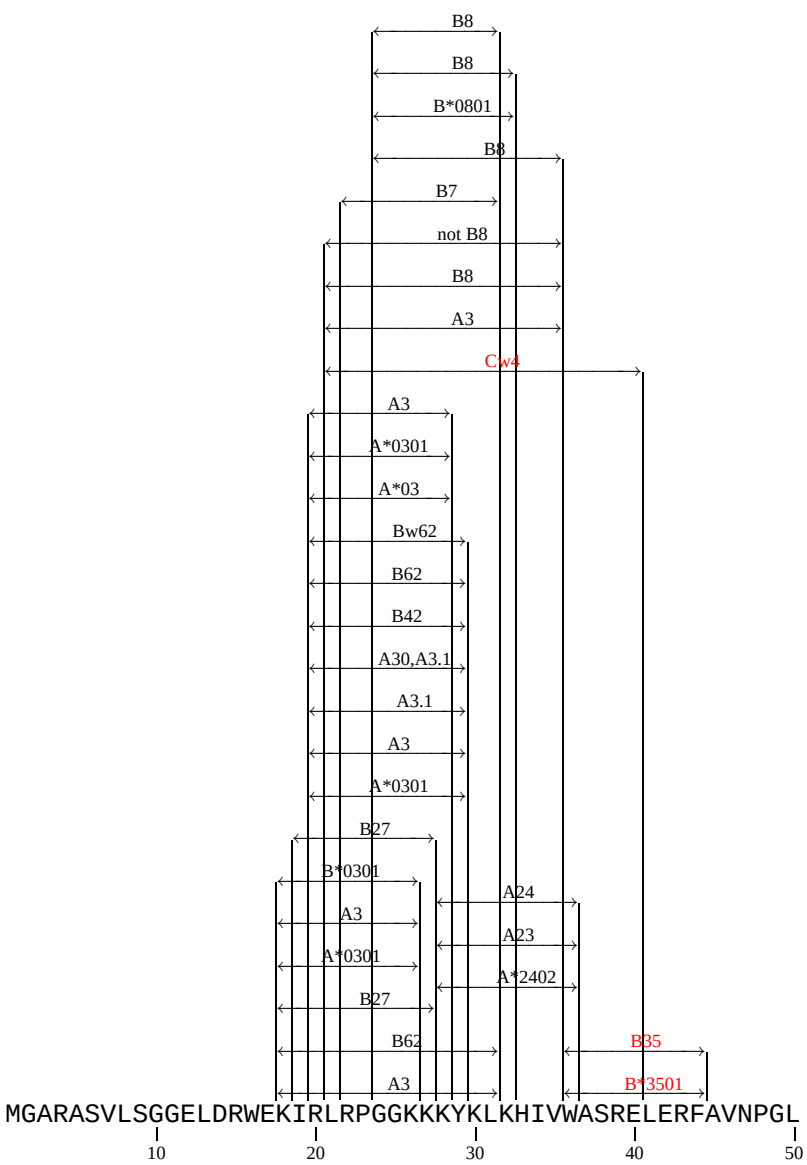
HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
RT(108–122)	RT(257–251)	VLDVGDAYFSVPLDE	HIV-1 infection	human(Cw4)	[Bernard (1998)]
	• This study focuses on six rare long-term survivor HIV-infected people who were infected for many years without exhibiting immune dysregulation – such immunologically normal HIV-infected (INHI) cases occur at a frequency between 0.1 and 1% in the infected population • No direct CTL were found in any of the six INHIs, but above background CTLp activity was founded in 3/6 INHIs				
RT(113–120)	Pol(268–275 SF2)	DAYFSVPL	HIV-1 infection	human(B*5101, B24)	[Tomiyama (1999)]
	• HLA-B27, -B51, and -B57 are associated with slow progression to AIDS, while HLA -B35, -B8, -B24 are associated with a rapid progression to AIDS (Nat. Med. 2:405, 1996;Lancet 22:1187, 1986;Hum Immunol 22:73, 1988;Hum Immunol 44:156, 1995) • 15% of Japanese populations carry HLA-B51 while HLA-B27 and -B57 are detected in less than 0.3% • Of the 172 HIV-1 peptides with HLA-B*5101 anchor residues, 33 bound to HLA-B*5101, seven of these peptides were reactive with CTL from 3 B*5101 positive individuals, and six were properly processed • Four of the six epitopes were highly conserved among B subtype sequences, DAYFSVPL is conserved				

Table 8: **All De£ned Epitopes within the 20mer, regardless of HLA type**

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
RT(107–115)	RT(262–270 IIIB)	TVLDVGDAY		(B*3501)	[Brander & Goulder(2001)]
	• C. Brander notes this is a B*3501 epitope				
RT(107–115)	RT(262–270 IIIB)	TVLDVGDAY	HIV-1 infection	human(B35)	[Wilson (1996), Menendez-Arias (1998)]
	• Epitope de£ned in the context of the Pediatric AIDS Foundation ARIEL Project, a mother-infant HIV transmission study • TVLDMGDAC is a naturally occurring variant that is less reactive • [Menendez-Arias (1998)], in a review, notes that this epitope includes a catalytic residue (Asp-110) in the active site of RT				
RT(107–115)	Pol(262–270 IIIB)	TVLDVGDAY	HIV-1 infection	human(B35)	[Wilson (1999)]
	• This study describes maternal CTL responses in the context of mother-to-infant transmission • Detection of CTL escape mutants in the mother was associated with transmission, but the CTL-susceptible forms of the virus tended to be found in infected infants • An additional variant that gave a positive CTL response: TVLDMGDAC				
RT(108–118)	RT(267–277)	VLDVGDAYFSV	*in vitro* stimulation	human(A*0201)	[van der Burg (1996)]
	• High dissociation rate, but immunogenic in primary CTL induction after repeated stimulations with peptide • CTL generated by *in vitro* stimulation of PBMC derived from uninfected individual				
RT(108–118)	RT(267–277)	VLDVGDAYFSV	HIV-1 infection	human(A2)	[Kundu (1998)]
	• Allogeneic dendritic cells (DCs) were obtained from HLA-identical siblings, pulsed with rgp160 MN or A2-restricted HIV-1 epitope peptides, and infused monthly into six HIV-infected patients • 1/6 showed increased env-speci£c CTL and increased lymphoproliferative responses, 2/6 showed increase only in proliferative responses, and 3/6 showed no change – pulsed DCs were well tolerated • VLDVGDAYFSV is a conserved HLA-A2 epitope included in this study – 4/6 patients had this sequence as their HIV direct sequence, but only one of these had a detectable CTL response – the other two had the sequences EEDVGDAYFSV and ELDVGDAYFSV and no detectable CTL response				
RT(108–118)	RT(267–277)	VLDVGDAYFSV	*in vitro* stimulation	human(A2)	[van der Burg (1995)]
	• Binds HLA-A*0201 – CTL generated by *in vitro* stimulation of PBMC from an HIV negative donor • VLDVGDAYFSV is in a functional domain				

HXB2 Location	Author Location	Sequence	Immunogen	Species(HLA)	References
RT(108–122)	RT(257–251)	VLDVGDAYFSVPLDE	HIV-1 infection	human(Cw4)	[Bernard (1998)]
	• This study focuses on six rare long-term survivor HIV-infected people who were infected for many years without exhibiting immune dysregulation – such immunologically normal HIV-infected (INHI) cases occur at a frequency between 0.1 and 1% in the infected population • No direct CTL were found in any of the six INHIs, but above background CTLp activity was founded in 3/6 INHIs				
RT(113–120)	Pol(268–275 SF2)	DAYFSVPL	HIV-1 infection	human(B*5101, B24)	[Tomiyama (1999)]
	• HLA-B27, -B51, and -B57 are associated with slow progression to AIDS, while HLA -B35, -B8, -B24 are associated with a rapid progression to AIDS (Nat. Med. 2:405, 1996;Lancet 22:1187, 1986;Hum Immunol 22:73, 1988;Hum Immunol 44:156, 1995) • 15% of Japanese populations carry HLA-B51 while HLA-B27 and -B57 are detected in less than 0.3% • Of the 172 HIV-1 peptides with HLA-B*5101 anchor residues, 33 bound to HLA-B*5101, seven of these peptides were reactive with CTL from 3 B*5101 positive individuals, and six were properly processed • Four of the six epitopes were highly conserved among B subtype sequences, DAYFSVPL is conserved				

p17 CTL Map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B8
B*0801
B8
B7
not B8
B8
A3
Cw4
A3
A*0301
A*03
Bw62
B62
B42
A30,A3.1
A3.1
A3
A*0301
B27
B*0301
A3
A*0301
B27
B62
A3
A24
A23
A*2402
B35
B*3501
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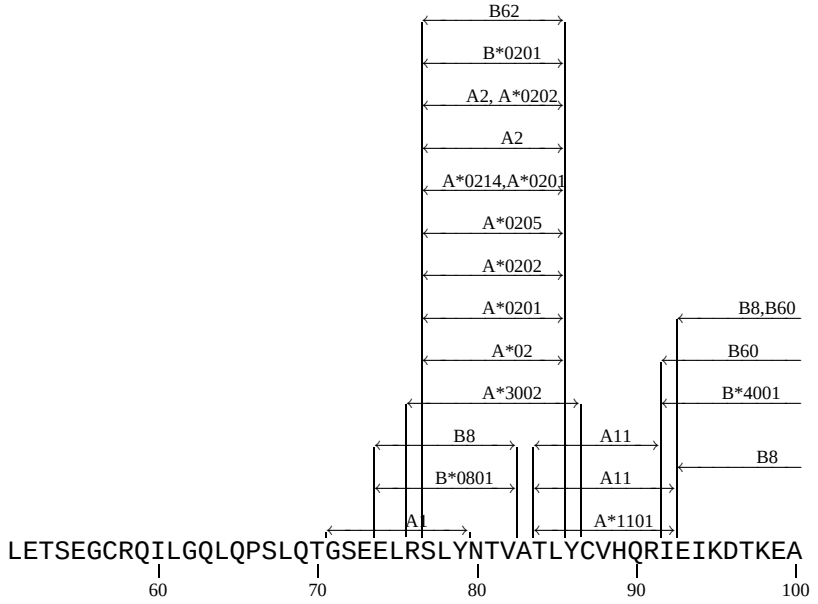
20
30
40
50


B62
B*0201
A2, A*0202
A2
A*0214,A*0201
A*0205
A*0202
A*0201
A*02
A*3002
B8
B*0801
A1
A11
A11
A*1101
B8,B60
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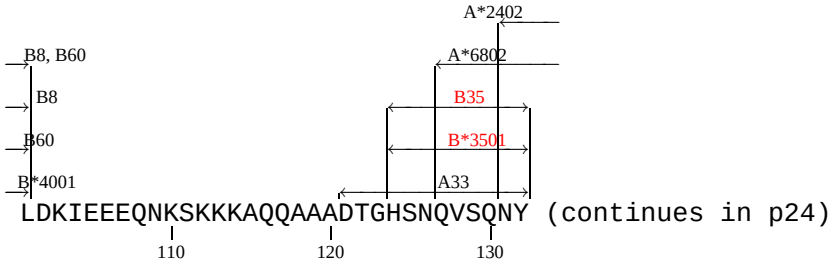
70
80
90
100


A*2402
A*6802
B35
B*3501
A33
B8, B60
B8
B60
B*4001
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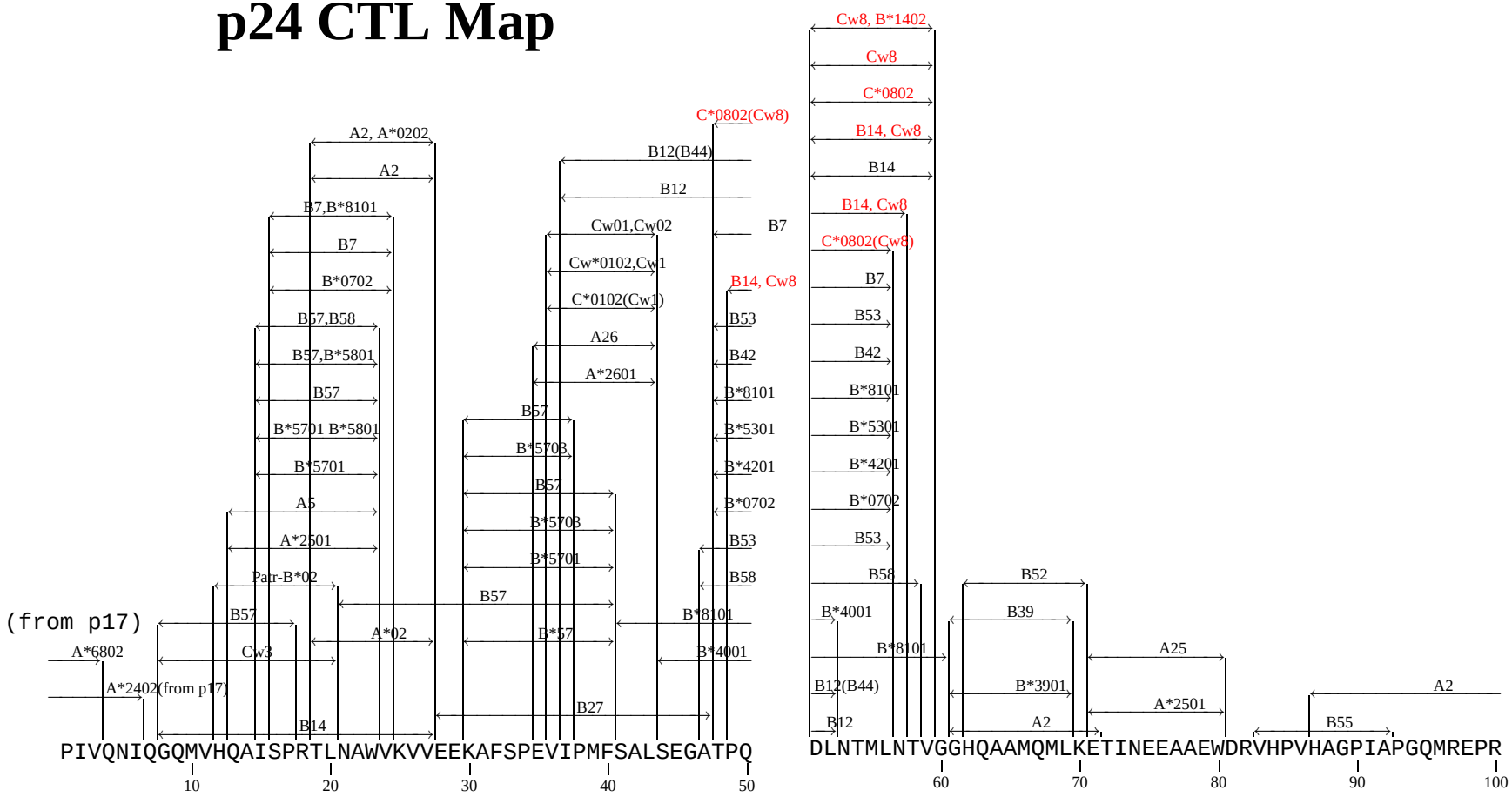
110
120
130

p24 CTL Map


(from p17)
A*6802
A*2402(from p17)
A2, A*0202
A2
B7,B*8101
B7
B*0702
B57,B58
B57,B*5801
B57
B*5701 B*5801
B*5701
A5
A*2501
Pair-B*02
B57
A*02
Cw3
B14
B12(B44)
B12
Cw01,Cw02
Cw*0102,Cw1
C*0102(Cw1)
A26
A*2601
B57
B*5703
B57
B*5703
B*5701
B57
B*57
B27
C*0802(Cw8)
B7
B14, Cw8
B53
B42
B*8101
B*5301
B*4201
B*0702
B53
B58
B*8101
B*4001
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10
20
30
40
50
Cw8, B*1402
Cw8
C*0802
B14, Cw8
B14
B14, Cw8
C*0802(Cw8)
B7
B53
B42
B*8101
B*5301
B*4201
B*0702
B53
B58
B*4001
B*8101
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B12
B52
B39
B*3901
A2
A25
A*2501
A2
B55
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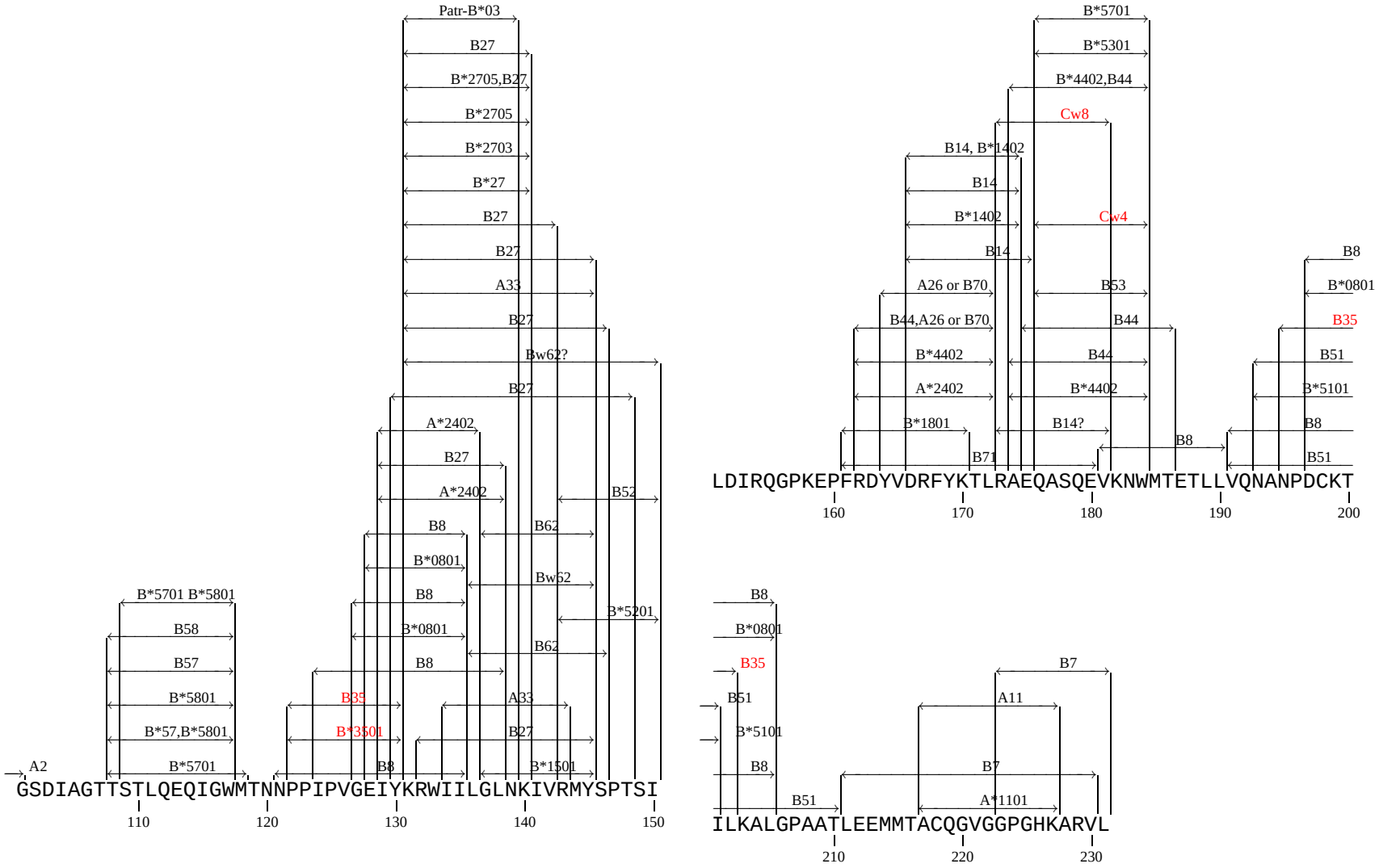
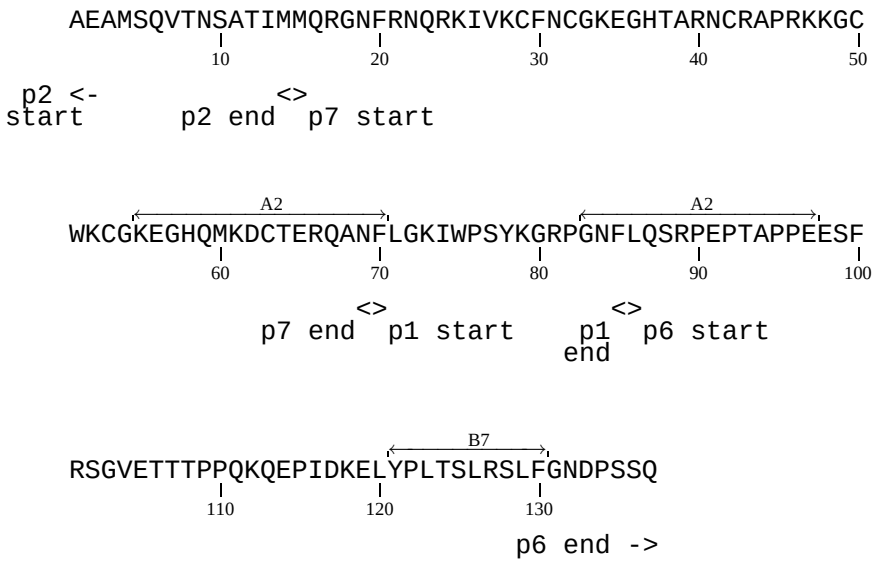
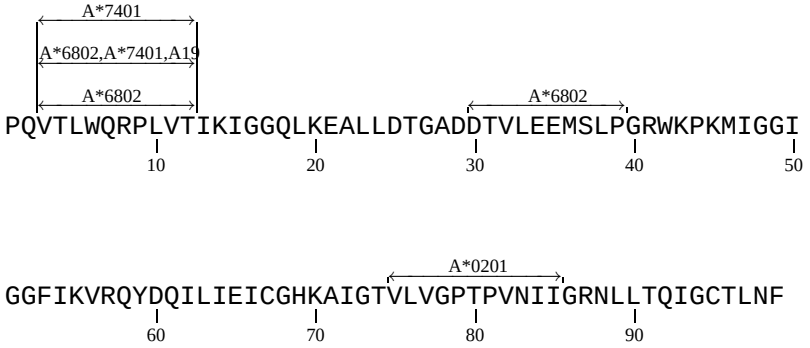
60
70
80
90
100


Patr-B*03
B27
B*2705,B27
B*2705
B*2703
B*27
B27
B27
A33
B27
Bw62?
B27
A*2402
B27
A*2402
B52
B8
B62
B*0801
Bw62
B*5701 B*5801
B8
B*5201
B58
B*0801
B62
B57
B8
B*5801
B35
A33
B*57,B*5801
B*3501
B27
A2
B*5701
B8
B*1501
GSDIAGTTSTLQEQIGWMTNNPPIPVGEIYKRWIILGLNKIVRMYSPTSI
110
120
130
140
150
B*5701
B*5301
B*4402,B44
Cw8
B14, B*1402
B14
B*1402
Cw4
B14
B8
A26 or B70
B53
B*0801
B44,A26 or B70
B44
B35
B*4402
B44
B51
A*2402
B*4402
B*5101
B*1801
B14?
B8
B8
B71
B51
LDIRQGPKEPFRDYVDRFYKTLRAEQASQEVKNWMTETLLVQNANPDCKT
160
170
180
190
200
B8
B*0801
B35
B51
B*5101
B8
B51
ILKALGPAATLEEMMTACQGVGGPGHKARVL
210
220
230
B7
A11
B7
A*1101

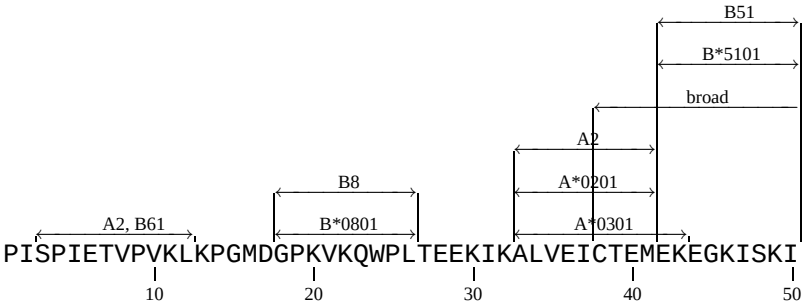
p2p7p1p6 CTL Map

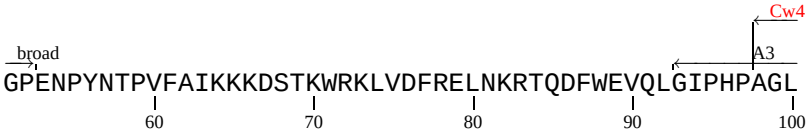


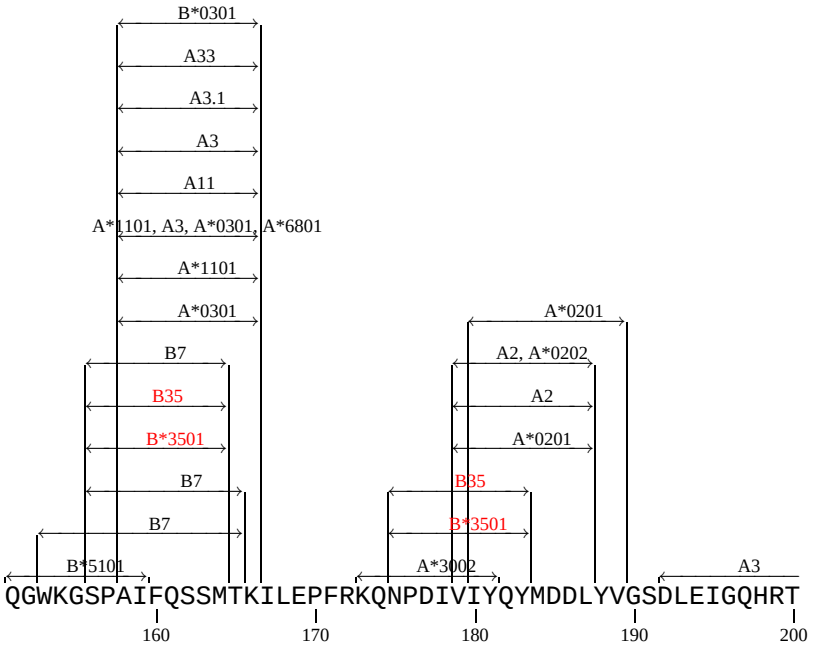
Protease CTL Map

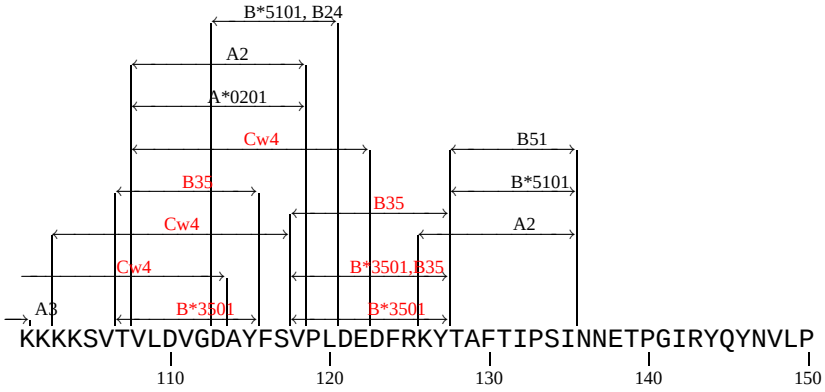


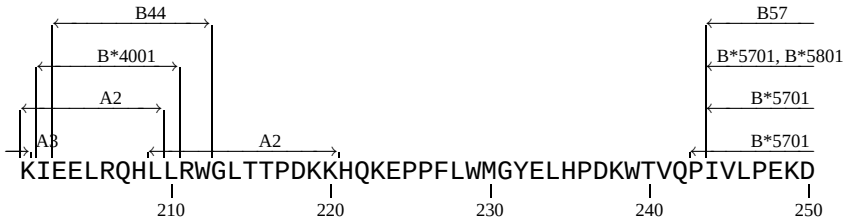
RT CTL Map

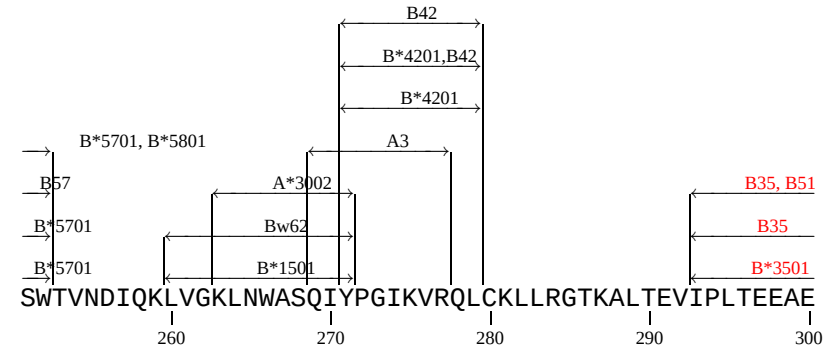

B51
B*5101
broad
A2
B8
A*0201
A2, B61
B*0801
A*0301
PISPIETVPVKLKPGMDGPKVKQWPLTEEKIKALVEICTEMEKEGKISKI
10
20
30
40
50

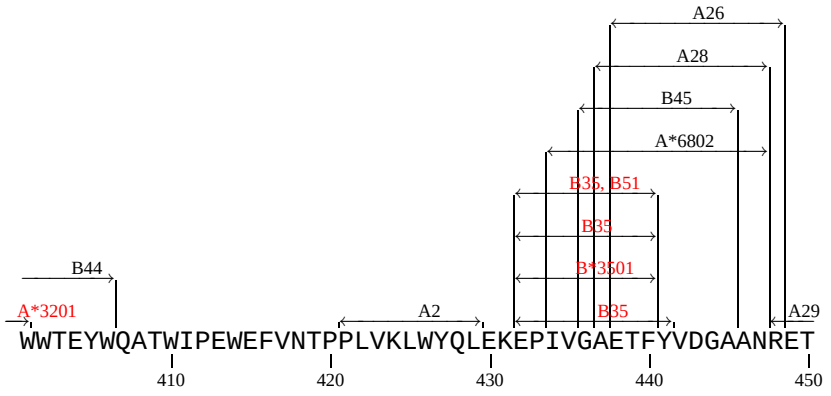

Cw4
broad
A3
GPENPYNTPVFAIKKKDSTKWRKLVDFRELNKRTQDFWEVQLGIPHPAGL
60
70
80
90
100


B*0301
A33
A3.1
A3
A11
A*1101, A3, A*0301, A*6801
A*1101
A*0301
B7
B35
B*3501
B7
B7
B*5101
A*0201
A2, A*0202
A2
A*0201
B35
B*3501
A*3002
A3
QGWKGSPAIFQSSMTKILEPFRKQNPDIVIYQYMDDLYVGSDLEIGQHRT
160
170
180
190
200

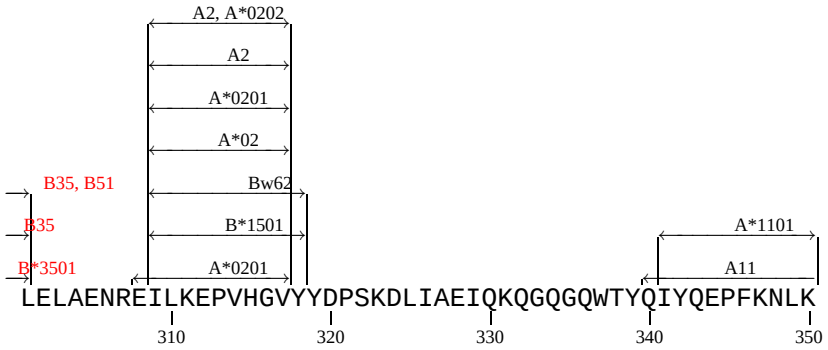

B*5101, B24
A2
A*0201
Cw4
B51
B35
B*5101
B35
Cw4
A2
Cw4
B*3501,B35
A3
B*3501
B*3501
KKKKSVTVLDVGDAYFSVPLDEDFRKYTAFTIPSINNETPGIRYQYNVLP
110
120
130
140
150

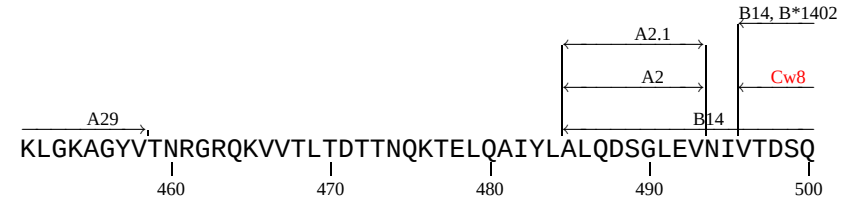

B44
B57
B*4001
B*5701, B*5801
A2
B*5701
A3
A2
B*5701
KIEELRQHLLRWGLTTPDKKHQKEPPFLWMGYELHPDKWTVQPIVLPEKD
210
220
230
240
250

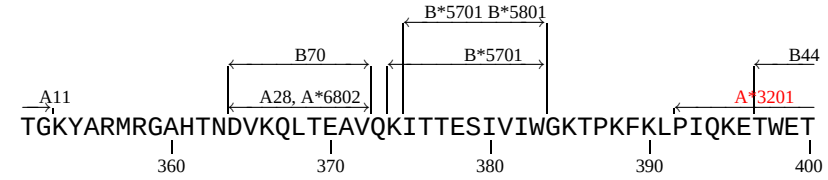


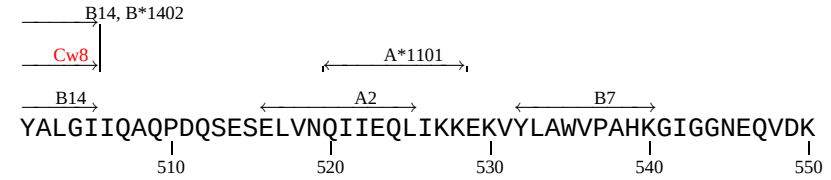


p15 RNase start <-





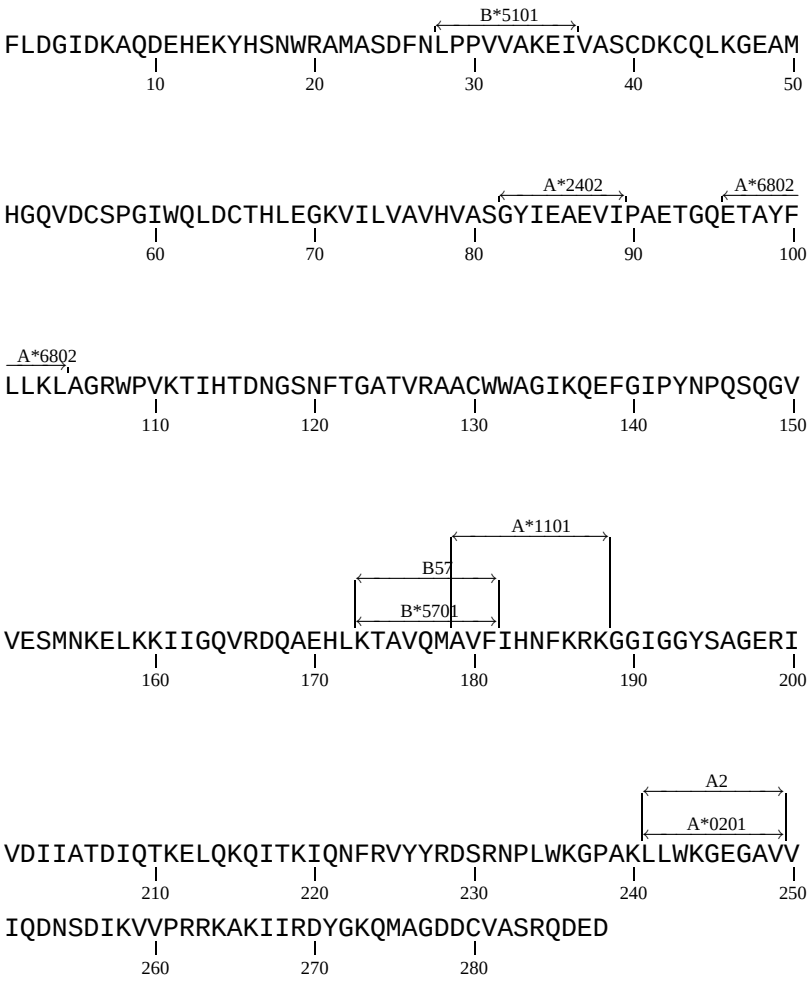




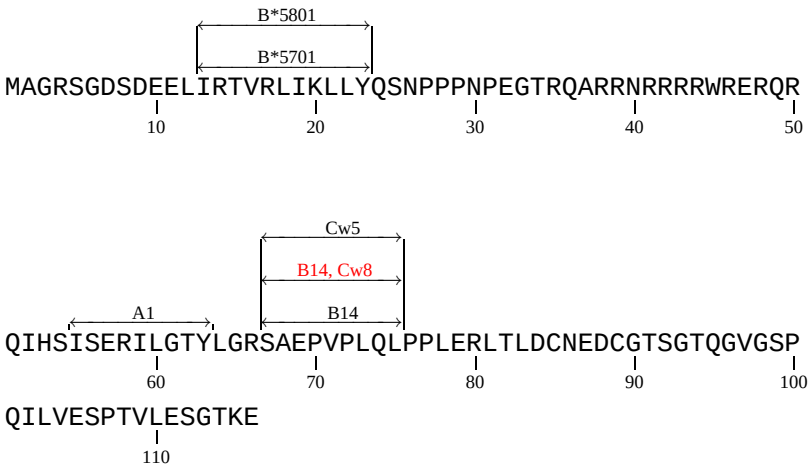
LVSAGIRKVL

-> p15 RNase end

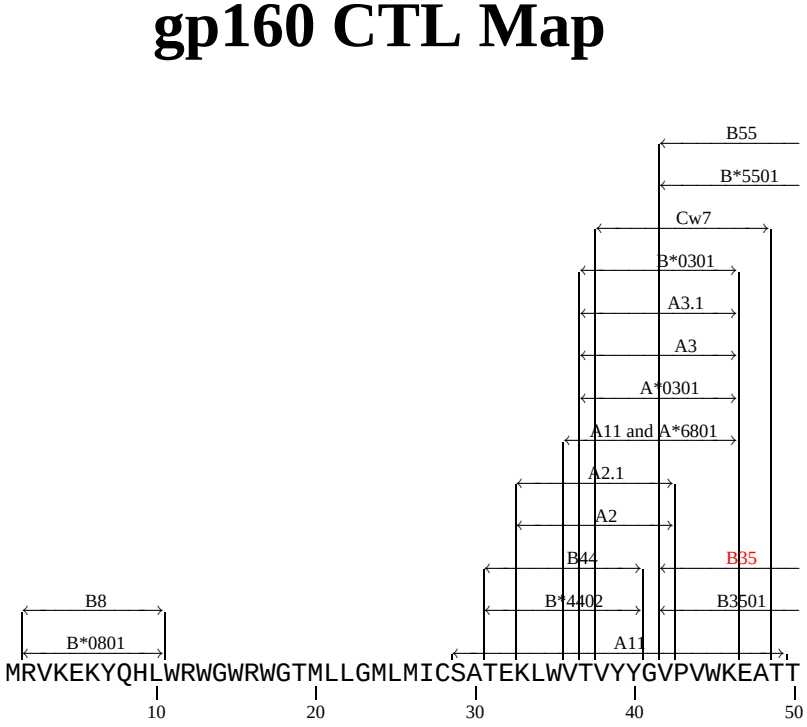
Integrase CTL Map



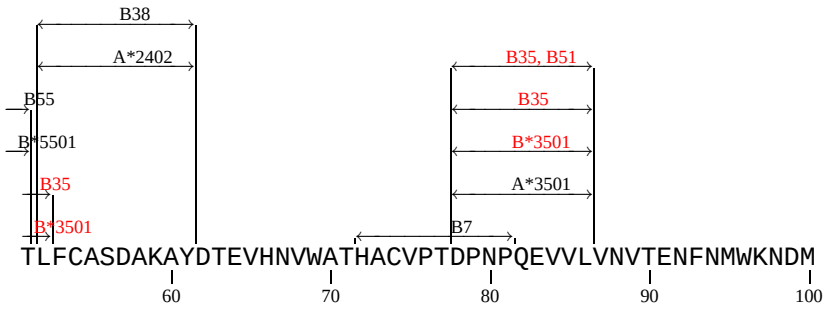
Rev CTL Map

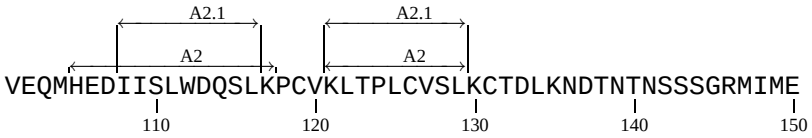


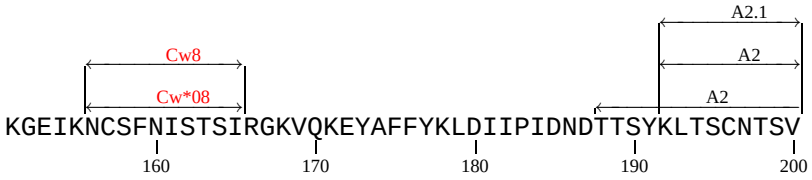
gp160 CTL Map

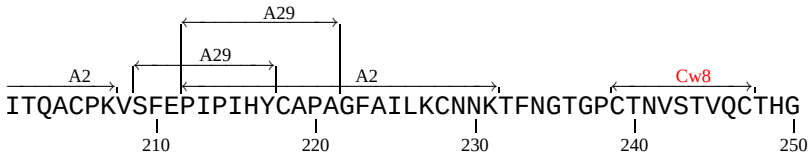


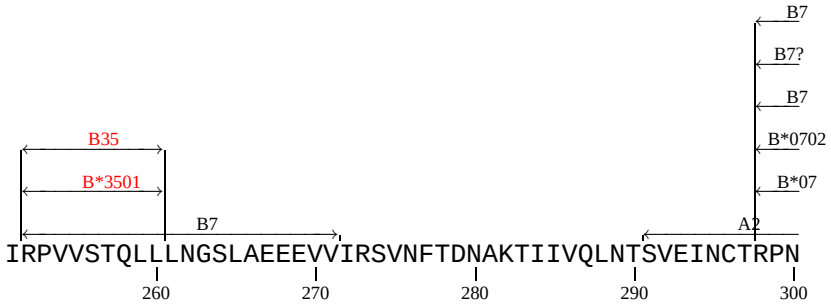
<- gp120 start

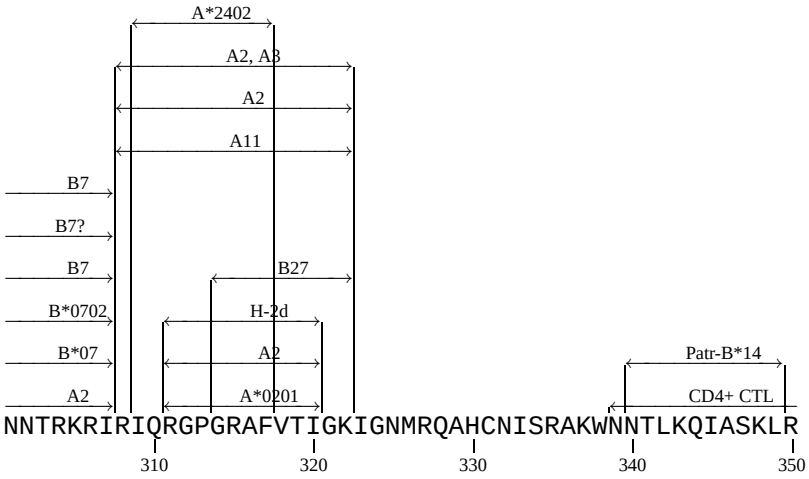


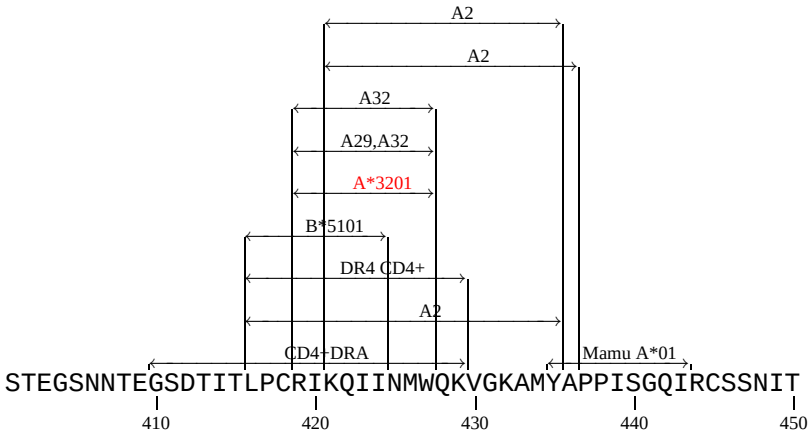


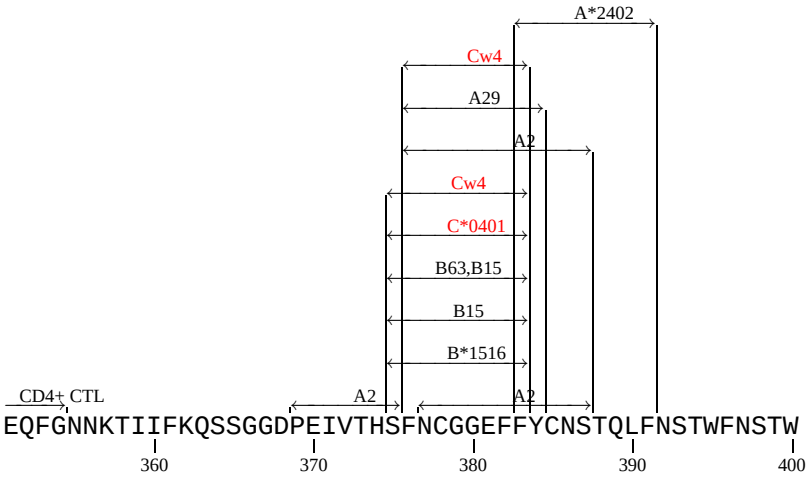




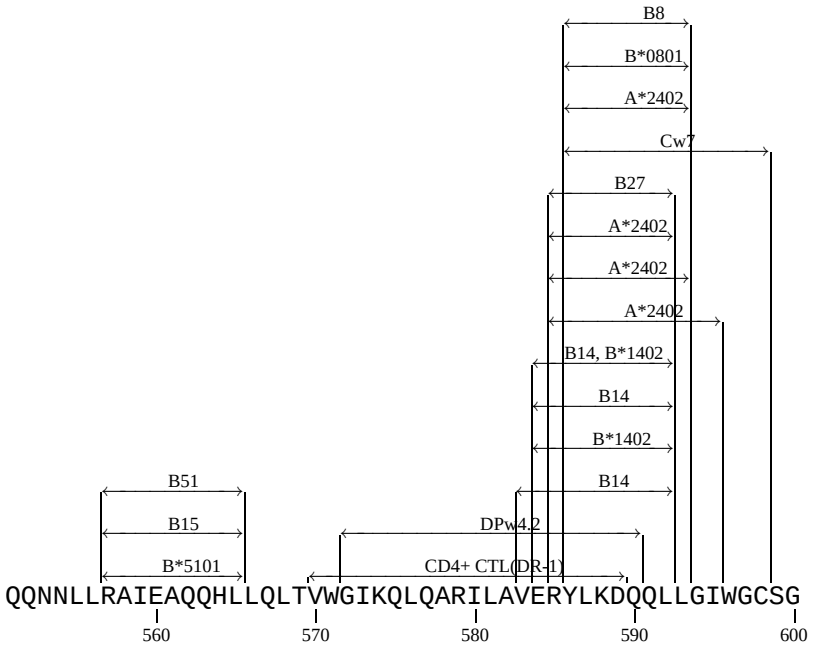


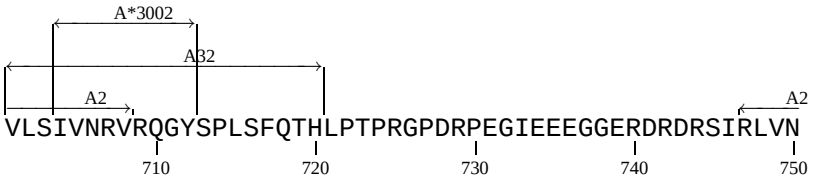


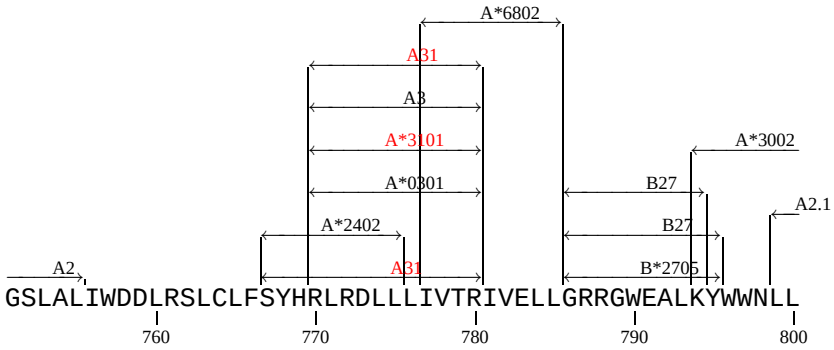




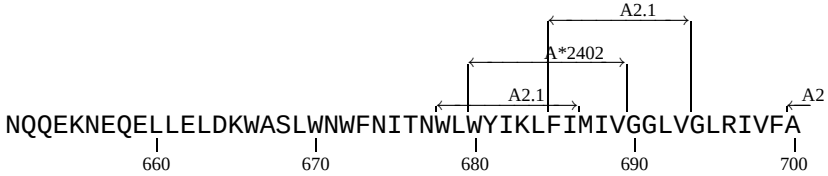
gp120 end <> gp41 start

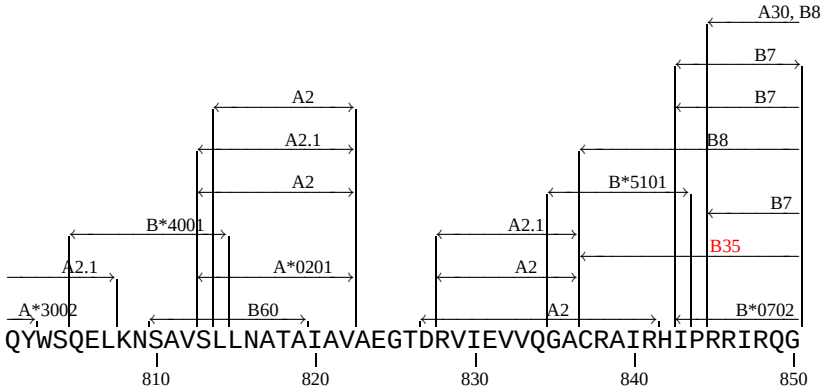

B8
B*0801
A*2402
Cw7
B27
A*2402
A*2402
A*2402
B14, B*1402
B14
B*1402
B14
B51
B15
B*5101
DPw4.2
CD4+ CTL(DR-1)
QQNNLLRAIEAQQHLLQLTVWGIKQLQARILAVERYLKDQQLLGIWGCSG
560
570
580
590
600

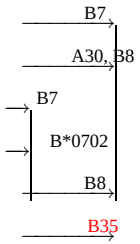

A*3002
A32
A2
A2
VLSIVNRVRQGYSPLSFQTHLPTPRGPDRPEGIEEEGGERDRDRSIRLVN
710
720
730
740
750


A*6802
A31
A3
A*3101
A*0301
A*2402
A31
A*3002
B27
B27
B*2705
A2.1
A2
GSLALIWDDLRSLCLFSYHRLRDLLLIVTRIVELLGRRGWEALKYWWNLL
760
770
780
790
800


B35
B*3501
KLICTTAVPWNASWSNKSLEQIWNHTTWMEWDREINNYTSLIHSLIEESQ
610
620
630
640
650


A2.1
A*2402
A2.1
A2
NQQEKNEQELLELDKWASLWNWFNITNWLWYIKLFIMIVGGLVGLRIVFA
660
670
680
690
700

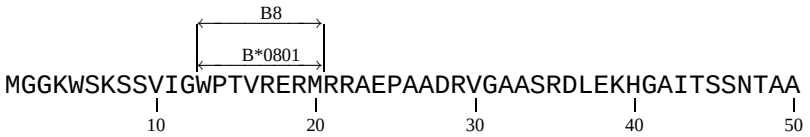

A30, B8
B7
B7
A2
A2.1
B8
A2
B*5101
B*4001
A2.1
B7
A2.1
B35
A*0201
A2
A*3002
B60
A2
B*0702
QYWSQELKNSAVSLLNATAIAVAEGTDRVIEVVQGACRAIRHIPRRIRQG
810
820
830
840
850

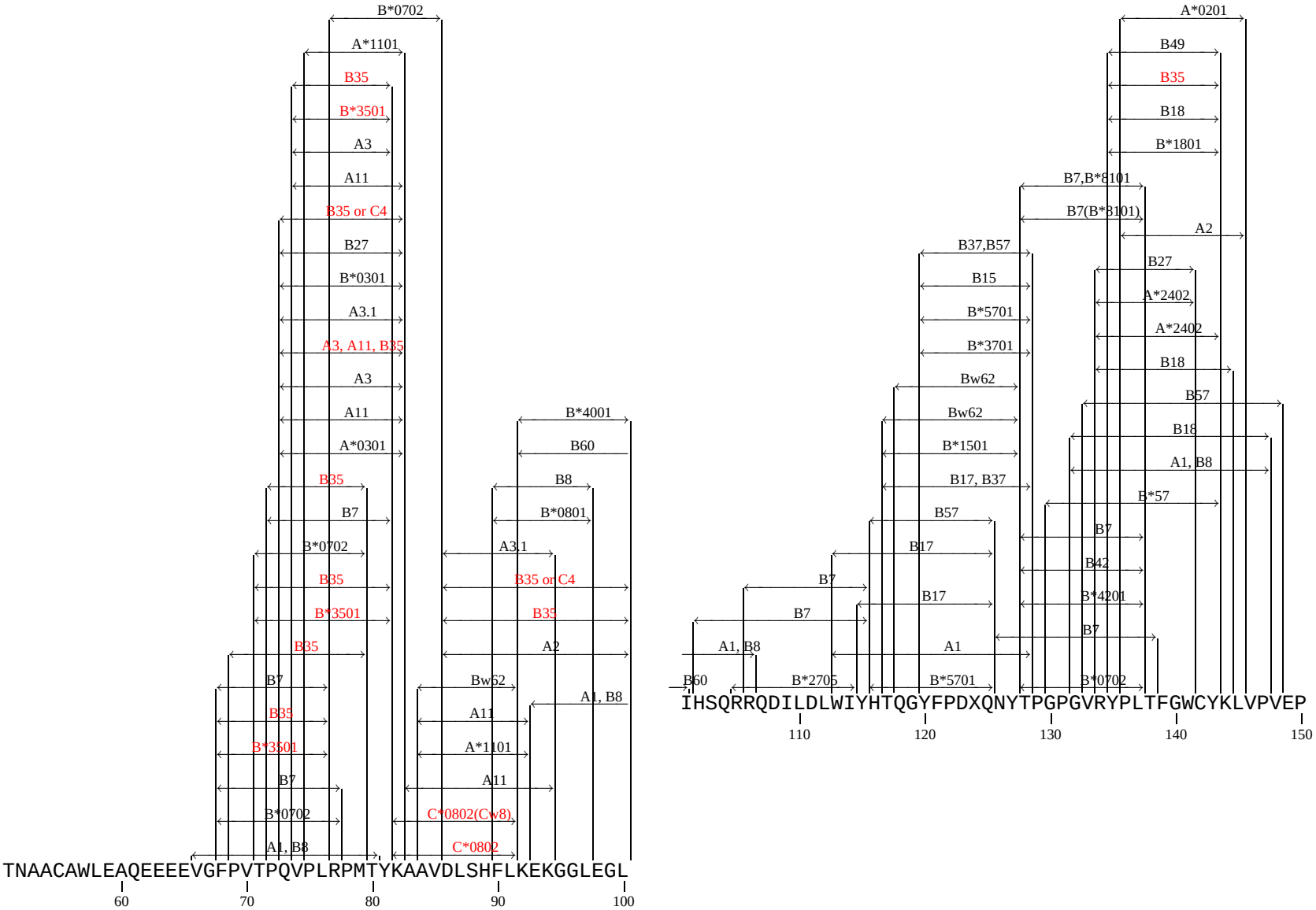


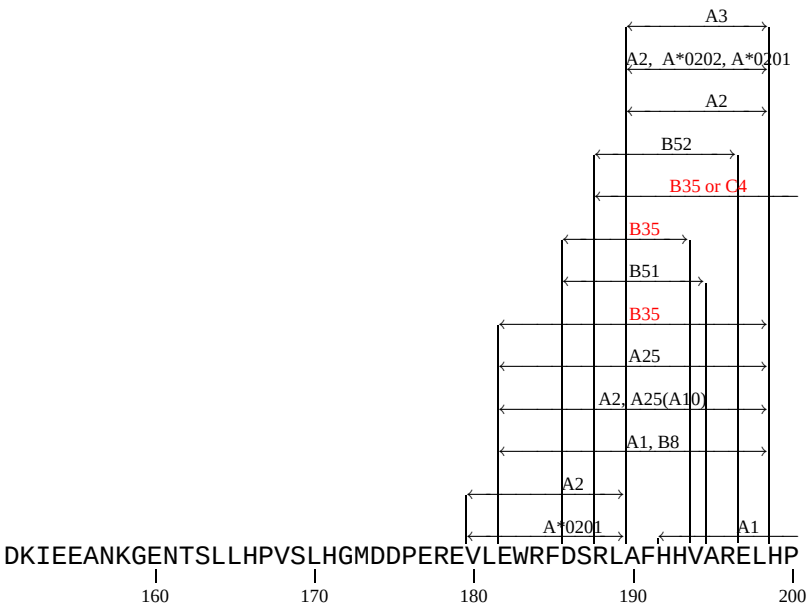
LERILL

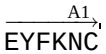
-> gp41 end

Nef CTL Map




B*0702
A*1101
B35
B*3501
A3
A11
B35 or C4
B27
B*0301
A3.1
A3, A11, B35
A3
A11
A*0301
B35
B7
B*0702
B35
B*3501
B35
B7
B35
B*3501
B7
B*0702
A1, B8
B*4001
B60
B8
B*0801
A3.1
B35 or C4
B35
A2
Bw62
A1, B8
A11
A*1101
A11
C*0802(Cw8)
C*0802
TNAACAWLEAQEEEEVGFPVTPQVPLRPMTYKAAVDLSHFLKEKGGLEGL
60
70
80
90
100
A*0201
B49
B35
B18
B*1801
B7,B*8101
B7(B*3101)
A2
B37,B57
B27
B15
A*2402
B*5701
A*2402
B*3701
B18
Bw62
B57
Bw62
B18
B*1501
A1, B8
B17, B37
B*57
B57
B7
B17
B42
B7
B17
B*4201
B7
B7
A1, B8
A1
B60
B*2705
B*5701
B*0702
IHSQRRQDILDLWIYHTQGYFPDXQNYTPGPGVRYPLTFGWCYKLVPVEP
110
120
130
140
150





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[Ferris (1999)] R. L. Ferris, C. Hall, N. V. Sipsas, J. T. Safrit, A. Trocha, R. A. Koup, R. P. Johnson, & R. F. Siliciano. Processing of HIV-1 envelope glycoprotein for class I-restricted recognition: dependence on TAP1/2 and mechanisms for cytosolic localization. *J Immunol* **162**:1324–32, 1999. (Medline: 99138809).

[Hammond (1995)] S. A. Hammond, R. P. Johnson, S. A. Kalams, B. D. Walker, M. Takiguchi, J. T. Safrit, R. A. Koup, & R. F. Siliciano. An epitope-selective transporter associated with antigen presentation TAP-1/2-independent pathway and a more general TAP-1/2-dependent antigen-processing pathway allow recognition of the HIV-1 envelope glycoprotein by CD8+ CTL. *J Immunol* **154**:6140–6156, 1995. (Medline: 95271010) Notes: Two peptide-processing pathways are utilized for MHC class I presentation of HIV-1 Env epitopes. The previously characterized TAP-1 and TAP-2 dependent pathway can generate all Env epitopes and uses Env protein mislocalized in the cytosol to produce peptides. The second, novel pathway uses a TAP-1/2 independent pathway, and allows a subset of MHC-restricted epitopes to be processed in the endoplasmic reticulum or a Golgi compartment.

[Harrer (1996)] T. Harrer, E. Harrer, S. A. Kalams, P. Barbosa, A. Trocha, R. P. Johnson, T. Elbeik, M. B. Feinberg, S. P. Buchbinder, & B. D. Walker. Cytotoxic T lymphocytes in asymptomatic long-term nonprogressing HIV-1 infection. Breadth and speci£city of the response and relation to in vivo viral quasispecies in a person with prolonged infection and low viral load. *J Immunol* **156**:2616–2623, 1996. (Medline: 96180222).

[Johnson (1994)] R. P. Johnson, S. A. Hammond, A. Trocha, R. F. Siliciano, & B. D. Walker. Induction of a major histocompatibility complex class I-restricted cytotoxic T-lymphocyte response to a highly conserved region of human immunode£ciency virus type 1 (HIV-1) gp120 in seronegative humans immunized with a candidate HIV-1 vaccine. *J Virol* **68**:3145–3153, 1994. (Medline: 94202302) Notes: In two volunteers, immunization with a single strain of HIV-1 induced CD4+ and CD8+ CTL that are speci£c for multiple conserved regions of HIV-1 and would be expected to recognize a broad range of viral isolates. The immunodominant gp120 epitope, gp120 TVYYGVPVWK, elicited CD8+ HLA-A3.1 restricted CTL, and this epitope is highly conserved. CTL speci£c for this epitope could lyse target cells sensitized with all known natural sequence variants. Additionally, CD8+ HLA-B35 and CD8+ HLA-B18 restricted epitopes were de£ned as well as two CD4+ cytotoxic T-cell gp120 epitopes: ITQACPKVSFEPIPHY-CAPAGFAI and NNTLKQIDSKLREQFG.

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[Safrit (1994b)] J. T. Safrit, A. Y. Lee, C. A. Andrews, & R. A. Koup. A region of the third variable loop of HIV-1 gp120 is recognized by HLA-B7-restricted CTLs from two acute seroconversion patients. *J Immunol* **153**:3822–3830, 1994b. (Medline: 95015873) Notes: HIV-1 envelope-speci£c CTL clones were isolated from the peripheral blood of two patients within weeks of seroconversion. These clones were CD8+ and restricted by the HLA-B7 molecule. The minimum epitope was de£ned, RPNNNTRKSI, with anchor residues at the proline and isoleucine; the anchor residues are relatively well conserved. A Serine to Arginine change at position 9 of the epitope abrogated clone recognition in one of the patients. This amino acid change is one factor that has been associated with a change from a nonsyncytium-inducing to a syncytium-inducing phenotype of HIV-1.

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M. Takiguchi. Identi£cation of multiple HIV-1 cytotoxic T cell epitopes presented by human leukocyte antigen B35 molecule. *AIDS* **10**:1075–1083, 1996. (Medline: 97028610).

[Sipsas (1997)] N. V. Sipsas, S. A. Kalams, A. Trocha, S. He, W. A. Blattner, B. D. Walker, & R. P. Johnson. Identi£cation of type-speci£c cytotoxic T lymphocyte responses to homologous viral proteins in laboratory workers accidentally infected with HIV-1. *J Clin Invest* **99**:752–62, 1997. (Medline: 97197584) Notes: To examine a situation where the autologous strain and the reference reagents would be the same, the CTL response of three lab workers accidentally infected with HIV IIIB was studied. Both group speci£c and type speci£c epitopes were targets for CTL clones. One subject had a broadening of CTL response over time, using a broad range of restricting HLA class I alleles. Characterization of the cytotoxic T lymphocyte (CTL) response against HIV-1 has been limited by the use of target cells expressing viral proteins from laboratory isolates of HIV-1. This approach has favored identi£cation of group-speci£c CTL responses and precluded assessment of the extent of type-speci£c CTL responses directed against HIV-1. Using cells expressing viral proteins from the HIV-1 IIIB strain, we performed a detailed characterization of HIV-1-speci£c CTL response in three laboratory workers accidentally infected with HIV-1 IIIB. Eight of the epitopes identi£ed were group speci£c, lying in relatively conserved regions of Gag, reverse transcriptase, and envelope. Three type-speci£c epitopes were identi£ed, two of them in highly variable regions of envelope. In longitudinal studies in one subject, seven different epitopes and £ve different restricting HLA class I alleles were identi£ed, with a progressive increase in the number of CTL epitopes recognized by this subject over time. Our data demonstrate that type-speci£c CTL responses make up a signi£cant proportion of the host cellular immune response against HIV-1 and that a broadening of epitope speci£city may occur.

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